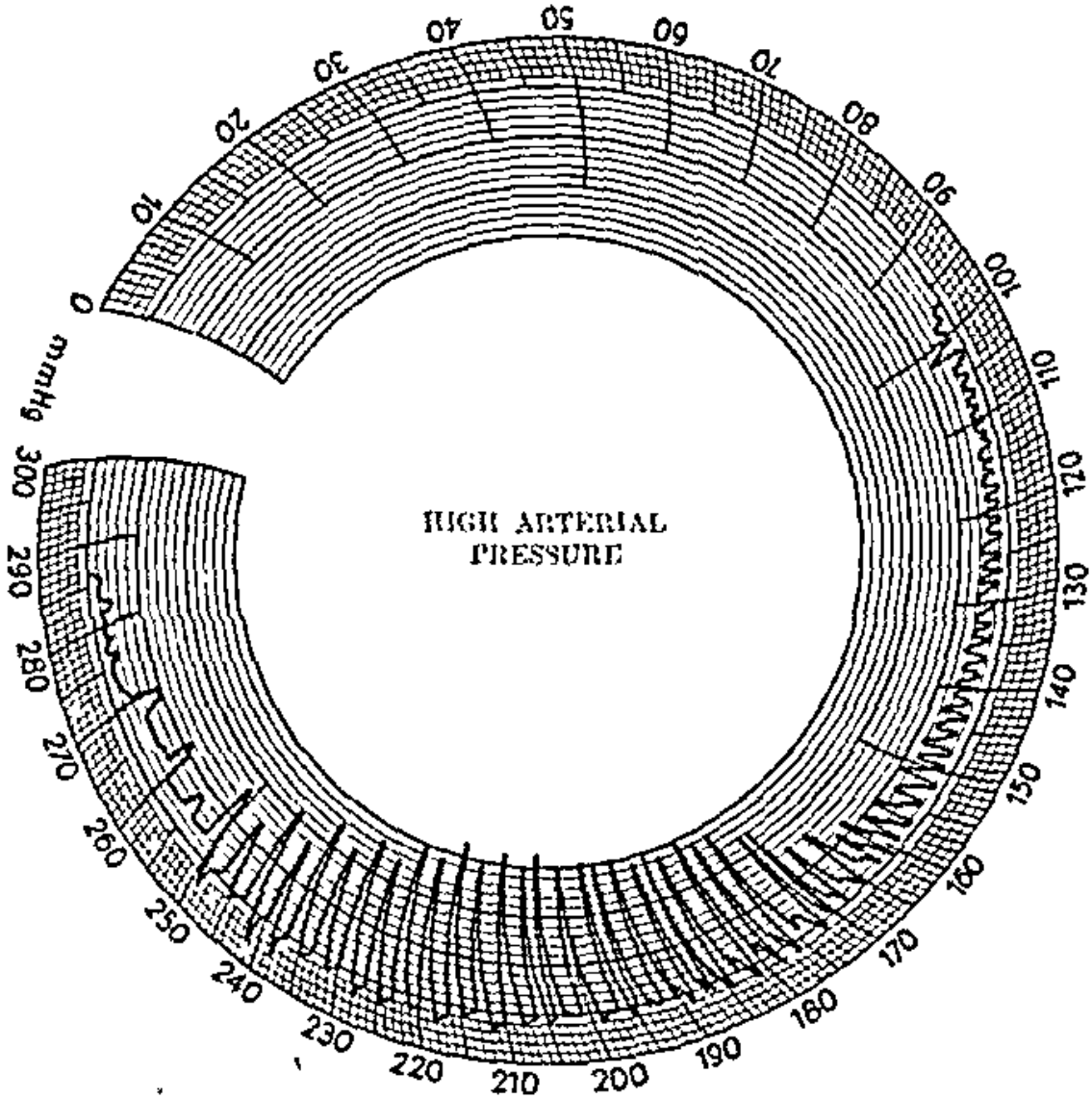

Frontispiece

GRAPHIC TRACING BY THE AUTHOR WITH THE TONOSCILLOGRAPH OF PTESCH showing high arterial pressure and *pulsus alternans* in a man aged 47. Systolic pressure is at 269 mm Hg., diastolic at 151 mm. Hg, and mean pressure at 201 mm Hg

HIGH BLOOD PRESSURE

ITS VARIATIONS AND CONTROL

A MANUAL FOR PRACTITIONERS

BY

J. F. HALLS DALLY

M.A., M.D., Cantab.,

MEMBER OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON
SENIOR PHYSICIAN TO THE MOUNT VERNON HOSPITAL AND TO THE ST. MARYLEBONE GENERAL DISPENSARY
DIRECTOR OF THE MARYLEBONE RHEUMATISM SUPERVISORY CENTRE FOR CHILDREN
CONSULTING PHYSICIAN TO THE SHRINERS' HOSPITAL, MONTREAL, AND MEMBER OF THE ADVISORY PATHOLOGICAL BOARD
CONSULTING PHYSICIAN TO THE PHILANTHROPIC SOCIETY'S SCHOOL, REDHILL

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T. CLIFFORD ALLBUTT

HOC OPUSCULUM

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LIBENTER DEDICAVIT

PREFACE TO THE THIRD EDITION

It is highly gratifying to the author that the continued welcome given to this book by practitioners, teachers and students of medicine justifies the appearance of a new edition.

In the endeavour to meet the needs of those for whom the book is intended, no efforts have been spared to render it simple, concise and adequate, the chief aim throughout having been to treat " High Blood Pressure " in a practical manner.

To this end, and in order to present the subject in readily assimilable form, although the main outlines remain the same, the Third Edition has been so largely rewritten as in many respects to constitute a new book.

As the result of present-day research and enlarged personal experience, salient points have been stressed, much relevant matter has been included, and much that was becoming out of date deleted. Twenty-four fresh illustrations have been added, and seven old ones replaced.

Careful revision has been made of sphygmomanometric methods and instruments, particularly those that are objective and self-registering.

The significance of mean arterial pressure has been discussed, and standard pressures at various ages have been revised to accord with the latest observations.

The section on hyperpiesia has been considerably amplified in the light of modern work. In like manner arteriosclerosis and its relations with hyperpiesis have been dealt with.

The causation, significance, varieties, symptomatology, associations and effects of high arterial pressure states have been extensively considered, while prognosis and end-results find appropriate mention.

My grateful thanks are due to numerous friends for valued help ; in particular to Dr. Fortescue Fox, who finds nothing

to modify in his useful contribution on " Baths and Waters," and to Dr. Knyvett Gordon, who has kindly revised for me the section on bacteriological and histological diagnosis. I am also indebted to the Editors of the *Lancet* and the *Medical Press and Circular* for permission to utilise respective articles from my pen on " Graphic Blood Pressure Records " and on " Milestones in the History of Blood Pressure " as bases for corresponding portions of Chapters III. and XV. ; to Messrs. Hawksley & Son, Short & Mason, Ltd., the W. A. Baum Co. Inc. (New York), Boulitte (Paris), and Professor J. Plesch (Berlin) for illustrations.

Last, but by no means least, I have to thank my publishers, Messrs. Wm. Heinemann (Medical Books) Ltd., for their courteous and ever-ready help.

J. F. H. D.

LONDON, W. 1.
July, 1934.

PREFACE TO THE SECOND EDITION

THE very cordial reception accorded by the profession to the first edition of "High Blood Pressure" appears to have justified its presentation as a text-book intended primarily for the use of general practitioners.

The Second Edition includes the most recent work on high arterial pressure and the diseases of which it is a symptom. The whole book has undergone careful revision: several chapters have been amplified, whilst much that was hazarded tentatively in the previous edition is now stated definitely on the ground of further considerable experience and personal observation. Many new illustrations, charts and tables have been added.

The terms "hyperpiesis," "hyperpiesia" and "hypertonia" are still so loosely employed that in Chapter V. the author has endeavoured to clarify the relationships of these terms by a new classification based on fundamental physical laws.

In Chapter X. will be found a series of observations on simultaneous brachial pressures in pulmonary tuberculosis.

Biochemistry, which has made such important strides in elucidating many vital processes hitherto but vaguely appreciated, is rapidly adding its quota towards the better understanding of changes in arterial pressure under conditions of disturbed metabolism. This subject, as well as various modern modes of treatment, has in the new edition received due notice.

The author's grateful acknowledgments are due to many friends: in particular, to Dr. R. Fortescue Fox for kindly contributing an Appendix to Chapter IX. on "The Treatment of Arterial Pressure by Baths and Waters"; to Dr. Henry Ellis for valuable help on the biochemical side and for useful suggestions on classification; to Dr. Sidney Bontor

and to Dr. A. F. Bill (Davos) for renewed assistance in the arduous task of proof-revision; to Dr. J. W. W. Adamson, Mr. H. L. Attwater and Mr. H. Robinson for various practical hints.

J. F. H. D.

London, W. 1,
June, 1926

PREFACE TO THE FIRST EDITION

WITHIN recent times blood pressure has become the object of widespread and increasing attention. The reasons for this are not far to seek. On the one hand, the modern physician is studying his patients with greater care, and as an aid to accurate diagnosis and treatment makes more frequent use of the sphygmomanometer ; on the other hand, public interest has been aroused to the importance of high arterial pressure by reason of the greater prevalence of the causes that induce it. In the words of Warfield, "there can be no doubt that arterial disease in the comparatively young is more frequent than it was twenty-five years ago, and that the mortality from diseases directly dependent on arteriosclerotic changes is increasing."

The exigencies of daily professional work preclude lengthy study of the extensive, scattered, and to some extent inconclusive literature which has grown up around the subject of blood pressure, especially of late years. As is only to be expected in a matter which admits of no absolute finality by the very nature of the human elements concerned, measurements cannot be stated in precise mathematical formulæ.

The aim of this manual is to present to the general practitioner in condensed and applicable form the modern views regarding blood pressure, and thus, in some instances, to supply a lack which has been present since his student days.

The book has been written in response to numerous requests from medical men actively engaged in the practice of their profession for a concise and handy volume in which they can readily find expressed in simple terms the essentials which they require to know.

Beginning, therefore, with a consideration of basic principles and technique, I have developed my thesis so as to emphasise the importance of the true index of diastolic pressure and the necessity of recording by the most suitable

method what I have termed "the complete arterial pressure picture," thus gradually leading up to a conception of abnormal arterial pressures as symptomatic of various underlying causes, certain of which are known, whilst others are as yet imperfectly understood. In the light of these premises, I have dealt with the control of high arterial pressure, bringing it into line with the preceding contents in the endeavour to render the conclusions based on personal experience a guide and help in daily practice.

By reason of their importance, arterial pressures in pulmonary tuberculosis and in relation to life assurance have received special notice in separate chapters. A review of the historical aspects of blood pressure estimation has been taken out of chronological order and relegated to the end of the book, so as not to break the sequence of more practical issues, but at the same time to render it available to those interested in the development of modern methods and technique.

In a volume of this size it has been thought well to avoid loading the text with notes of the mass of literature consulted. Should the reader desire further information touching various matters upon which considerations of space have forbidden me deeply to dwell, a list of the chief references is appended. In medicine there is no "always" and no "never." Hence I crave indulgence if the reasons for the faith that is in me are not, on every occasion, set forth in detail, or if, within the compass of these pages, individual mention of other writers to whom I am also indebted has not always been possible.

To colleagues and friends who have helped me with practical suggestions I tender my grateful thanks, in particular to Dr. Sidney Bontor for much sound advice and valuable criticism, and to Dr. Peter Miles for kindly reading through the proof-sheets.

J. F. HALLS DALLY.

93, Harley Street,
London, W. 1.

CONTENTS

PAGE

PREFACE vii

CHAPTER I

GENERAL CONSIDERATIONS. 1
Introductory 1
Reliability of Results of Clinical Blood Pressure Estimation 4

CHAPTER II

THE CLINICAL ESTIMATION OF ARTERIAL PRESSURE . . 6
When to take Blood Pressure 6
The Standard Measure of Arterial Pressure . . . 6
The Standard Width of the Compression Armlet . . 6
The Five Methods of Estimation:
1. The Auditory Method 7
The Auscultatory Arterial Pressure Phenomenon. 7
The Five Points and Phases of Sound in the Normal Auditory Curve 7
The Five Points of Sound. 9
Analysis of the Five Phases of Sound. . . 10
The Five Phases of Sound in Anomalous Auditory Curves 15
I. Variations in Intensity of Sounds produced in the Arterial Wall 15
II. Variations in Intensity of Sounds produced in the Blood Current 16
2. The Tactile Method 17
3. The Vibratory Method 20
4. The Oscillatory Method 21
5. The Graphic Method 21
Conclusions 22
The True Index of Diastolic Pressure 22
The True Index of Systolic Pressure 24
TECHNIQUE OF ARTERIAL PRESSURE ESTIMATION BY THE AUDITORY METHOD 25
The Complete Arterial Pressure Picture. . . . 27
Fallacies in Estimation of Arterial Pressure:—
A. Sources of Error affecting both Systolic and Diastolic Pressures 29
B. Sources of Error causing Over-estimation of the Systolic Pressure 29
(*a*) Factors External to the Artery under Compression 29
(*b*) Factors due to Variations in Resistance of the Arterial Wall 30

PAGE

Method of Relaxation of Contracted and Thickened Arteries to secure a Corrected Reading of Systolic Arterial Pressure 33
The Differential Blood Pressure Sign 33

CHAPTER III

Present day Instruments Employed in Estimation of Arterial Pressure 35
Blood Pressure Instruments to be recommended —
A. Mercurial Sphygmomanometers . . 35
The Baumanometer 36
B. Dial Sphygmomanometers (Aneroid) . . 40
1. The Improved Brunton Sphygmomanometer 40
2. The Boulitte Sphygmomanometer . . . 41
3. The Tycos Sphygmomanometer . 42
4 The Arteriotensiometer of Donzelot . . 44
C Oscillometric Sphygmomanometers (Aneroid) . 45
1. The Sphygmo-oscillometer of Pachon . 45
2. Boulitte's Universal Oscillometer . . . 49
3 The Kymometer of Vaquez, Gley and Gomez . 50
The Oscillometric Curve . 53
D. Self-recording Sphygmomanometers . . . 53
I. Indirect Clinical —
1. The Tonoscillograph of Plesch . . . 53
2. Boulitte's Portable Recording Oscillometer 61
3 Boulitte's Arterial Oscillograph . . . 62
4. The Tycos Recording Sphygmotonograph . 62
II. Direct Experimental —
The Boulittograph 65
Blood Pressure Instruments to be avoided . . 68

CHAPTER IV

Fundamental Physiological Factors in Blood Pressure 70
Significance of Blood Pressure 70
Minimal, Maximal and Mean Arterial Pressures . 74
Differential (Pulse) Pressure. 75
Mean Pressure . . . 75
The Aim of Blood Pressure 76
The Peripheral Resistance 77
The Sympathetic Vasomotor System . 77
Tone, Tension and Viscosity . . . 77

CHAPTER V

Physiological Variations in Arterial Pressure . . 81
Significance and Importance of the Diastolic Pressure . 81
Significance of the Systolic Pressure 85

PAGE
Significance of the Differential (Pulse) Pressure . . 85
Significance of the Mean Pressure 86
Average Normal Arterial Pressures 86
I. In Children 86
II. In Adults 88
Theoretical Standard Arterial Pressures at Various Ages 92
Simple Rules for calculating Arterial Pressures . . 93
Limits of Normal Arterial Pressure Variation . . . 94
Influence of Physiological Factors on Arterial Pressure . 94

CHAPTER VI

HYPERPIESIS; HYPERPIESIA; HYPERTONIA. 102
I. Hyperpiesis 102
II. Hyperpiesia 105
III. Hypertonia 113
Associations with Arteriosclerosis and Renal States . 114

CHAPTER VII

ARTERIOSCLEROSIS 117
Definition and Causation of Arteriosclerosis . . . 117
Pathology of Arteriosclerosis 119
Hyperpiesis and Arteriosclerosis 123

CHAPTER VIII

THE CAUSATION AND SIGNIFICANCE OF PATHOLOGICALLY HIGH ARTERIAL PRESSURE 125
Causal Factors 125
Hyperpiesis not necessarily a symptom of Cardio-arterio-renal Changes 136
Association of Hyperpiesis with Renal and Myocardial Involvement 137

CHAPTER IX

SYMPTOMATOLOGY OF HIGH ARTERIAL PRESSURE . . 140
A. Symptoms 140
B. Signs 142
C. Effects:—
1. On the Heart 142
2. On the Arteries and Arterioles 143
3. On the Kidneys 144
4. On the Brain 144

CHAPTER X

	PAGE
Diagnosis and Prognosis of High Arterial Pressure	145
A. Diagnosis of High Arterial Pressure	145
Clinical Examination of the Patient	145
B. Bacteriological and Histological Investigations	146
C. Biochemical Investigations :—	
1. Tests of Renal Efficiency	147
(*a*) Urine Tests	147
(*b*) Blood Tests	147
(α) Blood Urea Content	147
(β) Blood Calcium and Blood Phosphorus Estimations	148
D. Prognosis of High Arterial Pressure	148
1. Favourable Features	149
2. Unfavourable Features	149
Pathologically High Arterial Pressures	151
Range of High Arterial Pressures	151
Basal Metabolism in Hyperpiesis	152
Importance of the Heart and Systolic Pressure in Prognosis	152
Importance of the Diastolic and Mean Pressures in Diagnosis and Prognosis	153
Raised Arterial Pressure in association with :—	
1. Arteriosclerosis	155
2. Chronic Heart Muscle Insufficiency	155
3. Acute Infections	157
4. Acute Nephritis	157
5. Chronic Nephritis	158
6. Cerebral Angiospasm	159
7. Cerebral Hæmorrhage	159
8. Cerebral Thrombosis	161
9. Aortic Regurgitation	161
10. Hyperthyroidism	162
11. Bilateral Carotid Pulsation	162
12. Ocular Changes	163
Latency	165
Effect on Mortality of High Arterial Pressures	166

CHAPTER XI

Prevention of High Arterial Pressure	168
1. The Hours of Activity	168
2. The Hours of Rest	173

CHAPTER XII

Control of High Arterial Pressure	174
High Arterial Pressures at times protective or compensatory	175

PAGE

Control varies in the following groups :—
1. Simple High Pressure Group 175
Psychical Influences 177
Angiospasm 177
Endocrine Influences 178
Hyperpiesia and Asthma 179
Climacteric Hyperpiesia 180
Hyperpiesis and Toxæmias of Pregnancy . . 181
Hyperpiesis and Fibroid Disease of the Uterus . 181
Hyperpiesis, Gout, Cardiac Œdema and Obesity . 182
Plethoric Hyperpiesis 182
2. Cardiovascular Group 183
A. The Heart 183
B. The Arteries 184
Control of Hyperpietic Cerebral Attacks . . 185
Control of Cerebral Hæmorrhage . . . 185
3. Renal Group 186
Control of Acute and Chronic Nephritis . . 186
Control of Hæmorrhages 189
Control of Uræmia 189
Control of Eclampsia and Pre-eclamptic States . 190
Psychical Measures 191
Diet 192
Physical Measures :—
1. Rest 196
2. Exercise 197
3. Deep Breathing Exercises 198
4. Relaxation Exercises and Passive Movements . . 199
5. Resistance Exercises 200
6. Massage 200
7. Diaphoresis 201
8. High Frequency and Diathermic Currents . . 201
9. Ultra-Violet Rays 203
10. X-Rays 203
11. Lumbar Puncture 203
12. Venesection and Fasting 204
13. Drug Therapy :—
Detoxication Treatment of Intestinal Toxæmia . 205
Purgatives 205
Intestinal Antiseptics 207
The Nitrite Group 208
Vasodilators 208
The Halogens 210
The Chloride Group 210
The Iodide Group 211
The Bromide Group 212
Other Nerve Sedatives 212
The Sulphocyanates, Bismuth 212
Subnitrate, Misletoe 213
Alkalis and Stomachics 213
Acids 213
Cardiac Tonics 217

13. Drug Therapy—*continued.* PAGE
Organotherapy :—
Pancreatic Extracts 214
Vagotonine 214
Angioxyl 216
Kallikrein 216
Insulin 216
Substances derived from Various Sources . . 216
Choline Derivatives 216
Octylic Alcohol 217
Hepatic Extracts 218
APPENDIX. The Treatment of High Arterial Pressure by Baths and Waters, by R. Fortescue Fox, M.D., F.R.C.P. 219

CHAPTER XIII

ARTERIAL PRESSURE IN PULMONARY TUBERCULOSIS . . 225
Low Arterial Pressures not the Rule in Chronic Pulmonary Tuberculosis 225
Decline in Arterial Pressures depends on Absorption of Toxins 226
Fluctuations with Altitude 227
Prognosis 227
The Author's Observations on Simultaneous Brachial Pressures in Pulmonary Tuberculosis . . . 228
Effect of Tuberculous Toxins upon Nervous and Endocrine Systems 231
Diminution in Inspiratory Descent of Diaphragm in Pulmonary Tuberculosis and its Effects . . . 232
Results of Treatment on Arterial Pressure in Tuberculosis 232

CHAPTER XIV

ARTERIAL PRESSURE IN RELATION TO LIFE ASSURANCE . 233
Indications afforded by the Diastolic Pressure . . 234
Importance of recording the Complete Arterial Pressure Picture 234
Value of Serial Estimations of Arterial Pressure . . 235
Effects of Associated Conditions on Arterial Pressure . 236
General Hints on Life Assurance Examination . . 237
Value of Sphygmomanometry in Life Assurance . . 238

CHAPTER XV

EPOCHS IN THE CLINICAL ESTIMATION OF ARTERIAL PRESSURE 240
1. The Circulation of the Blood 240
2. Direct Estimation of Arterial Pressure—Physiological :—
The Mercurial Manometer 242
The Kymograph 243
The Elastic or Spring Manometer . . . 243

HIGH BLOOD PRESSURE
ITS VARIATIONS AND CONTROL

CHAPTER I

General Considerations

"Life is short; art is long; experience is fallacious, and judgment difficult."
HIPPOCRATES: *Aphorism I.*

"Every man's own reason is his best Œdipus, and will upon a reasonable truce find a way to loose those bonds wherewith the subtleties of error have enchained our more flexible and tender judgment."
SIR THOMAS BROWNE: *Religio Medici.*

MEDICINE is an art, and, since the human factor precludes reduction of clinical data to exact mathematical formulæ, the use of a single method offers no royal road to success.

The fundamental characters of the circulation are still acknowledged to be in the main identical with those portrayed by the immortal Harvey,[1] to whom, incidentally, the capillary circulation was unknown, yet, particularly during the past half-century, detailed investigation, aided by the employment of instrumental methods, has had the effect of modifying many of our previous concepts. Among the appliances which aim at a higher degree of precision than can be attained by the unaided senses, however specialised, of the physician, the sphygmomanometer has met with a large and increasing measure of recognition.

The following pages constitute an attempt to indicate the position of sphygmomanometry in clinical medicine, and at the same time to assess both its advantages and its limitations. A good servant but a bad master, its findings must ever be viewed in their due perspective. To disdain, however, to profit by its aid, no longer accords with the prestige of a self-respecting practitioner of the healing art.

Present-day clinical estimations of arterial pressure in man

Thus digital investigation is inadequate. Further, it may even be most misleading in cases where the pulse is "small," where hypertonia is present, or where the limb is adipose. In short, for a large proportion of cases palpation of the pulse will afford no more exact information than that yielded, in the absence of a clinical thermometer, by a conjecture as to the height of a patient's temperature by the feel of the skin. To measure arterial pressure takes less time and trouble than to test the urine, and often proves of equal or greater value.

It is now generally realised that departures from physiological blood pressure standards are symptomatic of some underlying condition.

As regards transitory variations, abundant evidence is forthcoming that these constitute on the part of the organism an expression of reaction capable of modification within a wide normal range to meet the varying physiological needs of daily life.

Apart from such simple and well-recognised causes, we have to consider variations in blood pressure, whether temporary or permanent, which arise from pathological conditions the primary nature of which cannot always be fully determined. In like manner, these deviations are symptomatic of the underlying cause or causes upon due appreciation of which successful management must depend.

The pressor effect of supernormal arterial pressure would appear to be exerted primarily upon the central nervous system rather than upon the peripheral circulation. Impaired blood supply to the vasomotor centre in the medulla is admitted as a cause, but in many cases cannot be ascribed to arteriosclerosis of the vessels supplying the centre. The pathological cause, as suggested by Cushing, is of pituitary origin, and the primary causes inducing these effects, at any rate as regards hyperpiesia, are probably psychical or toxæmic.

Hence we are forced to abandon the older view that abnormal blood pressure of itself is a state which necessarily calls for drastic treatment by drugs in order either to raise the pressure when unduly low or to lower it when unduly

high, for if the nature of the cause be not understood, efforts on these lines will not only produce no improvement in the patient's health, but, on the contrary, are apt to induce subjective discomfort, if not indeed actual harm, even to the extent of culmination in a fatal issue.

In control of abnormal pressure states, the best prospect of relief to the patient is derived from close co-operation between the medical attendant and the consultant. In many cases improvement is necessarily gradual, so that careful assessment and revision of the effects of therapy at definite intervals are advisable.

Are the Results of Clinical Blood Pressure Estimation accurate?

Clinical estimations of blood pressure are approximate because they are indirect, and should not be regarded as exact in the strict mathematical sense. Nevertheless, thanks to the increasing perfection of present-day technique, it is now possible to determine both diastolic and systolic pressures with equal facility and accuracy, so that the results are almost as exact as those obtained in laboratory experiments by direct measurement, and for clinical purposes may be regarded as perfectly reliable.[3, 4, 5, 6, 7]

Carefully recorded blood pressure readings furnish us with information which is often of the greatest practical help, and, as personal experience grows, their value and importance become increasingly evident. Not only is this true for circulatory disturbances, but for many general diseases in the elucidation of which the sphygmomanometer is often of the greatest assistance and will save the practitioner from making many mistakes.

Being satisfied that the present-day methods of estimating arterial pressure are reliable, in the next place it is necessary to consider the value and significance of the results obtained. Properly to assess these we must know :—

1. The best practical method of taking arterial pressures (Chapter II.).

2. The fundamental factors in their causation and maintenance (Chapter IV.).

3. The physiological limits within which arterial pressures may vary (Chapter V.).

4. The causes and significations of departures from the physiological standards for age and weight (Chapters VIII., IX. and X.).

5. The nature of the steps to be taken when necessary to control such departures (Chapters XI. and XII.).

CHAPTER II

The Clinical Estimation of Blood Pressure

"I regard the measurement of blood pressure as the most important of all the resources that have been added to our armamentarium in the last fifteen years." Cabot, 1913.

When should Blood Pressure be taken ?

1. At the first examination as a guide to diagnosis, prognosis and treatment

2. Subsequently at intervals to register progress and to ascertain the value of certain lines of management, particularly in (*a*) cardiovascular disturbances, (*b*) renal disease; (*c*) hyperthyroidism, (*d*) pulmonary affections, especially tuberculosis; (*e*) psychical states; (*f*) cerebral and abdominal injuries and diseases; (*g*) obstetrics, including eclampsia, and gynæcology; (*h*) anæsthesia and surgery, before, during and after operation, (*i*) intravenous injections for syphilis.

3. At any examination involving physical or mental fitness.

4. All subjects above middle age should be encouraged to present themselves for sphygmomanometric investigation at least once yearly, since one is thus enabled to discover when their arterial pressure is beginning to mount beyond standard limits, and to adopt measures of precaution by which in the majority of cases life can probably be prolonged

The Standard Measure of Arterial Pressure

The generally accepted standard is the height in millimetres of a column of mercury, or an equivalent measure in instruments calibrated from that source.

The Standard Width of the Compression Armlet

The distensible elastic rubber cuff, encircling the arm and surrounded by an external inexpansible fabric, should have

a width of not less than 12 cm. when used for auditory determination of arterial pressure in adults, and of 9 cm. for children. If the cuff be narrower than the standard width, all readings will be too high. For infants and small children it is often convenient to measure the pressure in the femoral artery.

THE FIVE METHODS OF ESTIMATING ARTERIAL PRESSURE

All the methods of estimating arterial pressure fall under five main heads .—

1. The Auditory Method.
2. The Tactile Method.
3. The Vibratory Method.
4. The Oscillatory Method.
5. The Graphic Method.

1. The Auditory or Auscultatory Method

Since 1905, when Korotkow [8] suggested the estimation of arterial pressure by auscultation, this method, by reason of its simplicity, quickness and accuracy, has come to be largely adopted as a routine measure.

The Auscultatory Arterial Pressure Phenomenon.—On placing the bell of a binaural stethoscope (or an auditory tambour) over the brachial artery at the bend of the elbow and just below the zone of compression exerted by a circular pneumatic armlet, on gradually lowering the pressure within the armlet, a series of sounds becomes audible. These sounds, while presenting similar general characteristics, nevertheless, in individual cases, show variations from which useful deductions in respect of diagnosis may be drawn.

THE FIVE POINTS AND PHASES OF SOUND IN THE NORMAL AUDITORY CURVE

At certain points the character of sounds changes, the duration of successive zones of sound between any two successive points, as measured in millimetres of mercury, being termed phases.

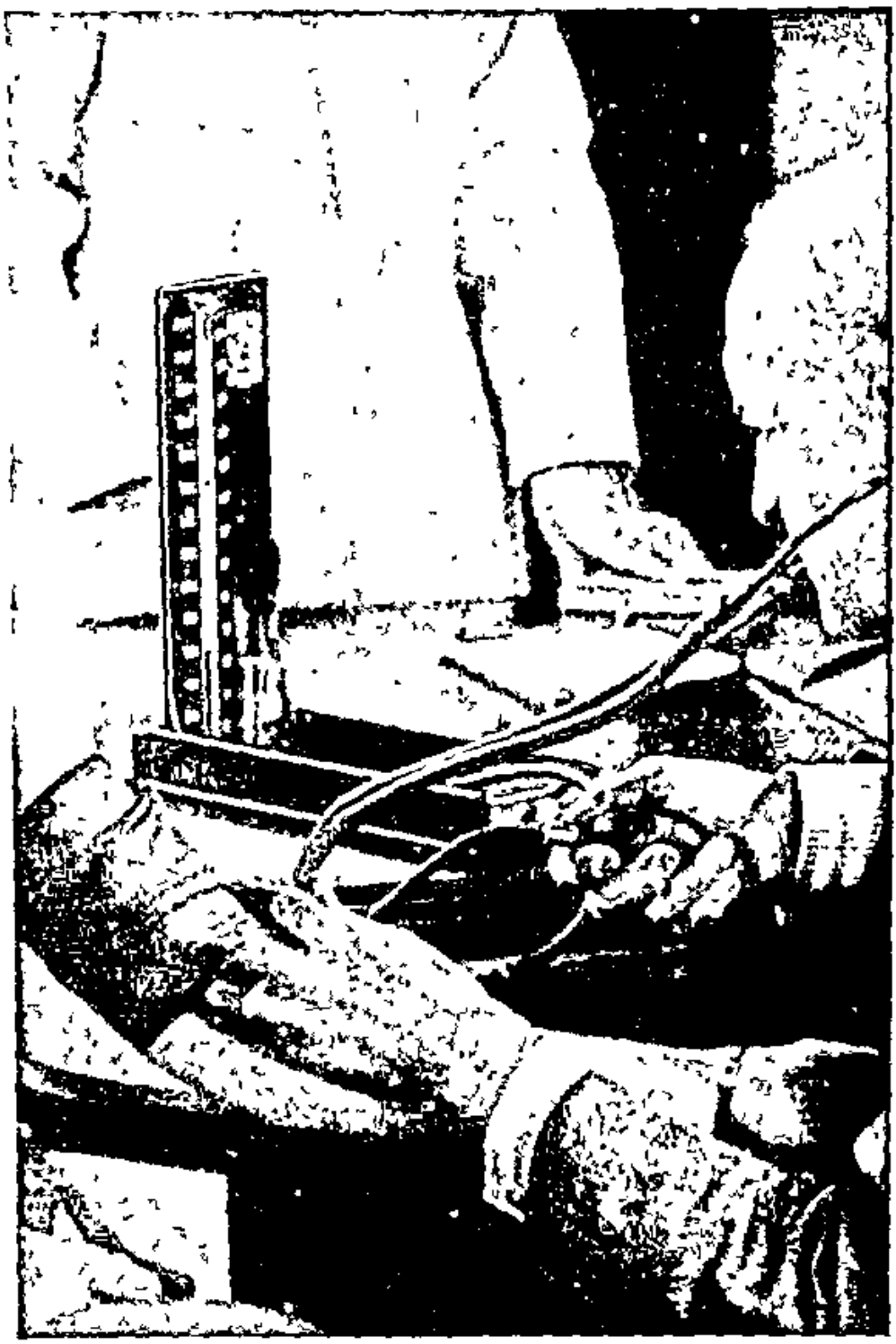


Fig. 1.—The auditory method of estimation of arterial pressure (pp. 7-17 and 25-29). The instrument figured is the latest form of desk model Baumanometer, which reads from 0 to 300 mm., and is the one most generally applicable to consulting room and hospital work. Size 2½ × 4½ × 13½ in.

As the armlet pressure is lowered, the five phases of sound [9] (Fig. 2) occur in the following order :—

1. Clicks.
2. Murmurs.
3. Thuds.
4. Dull Sounds.
5. Silence.

The Auditory Sequence of Sounds

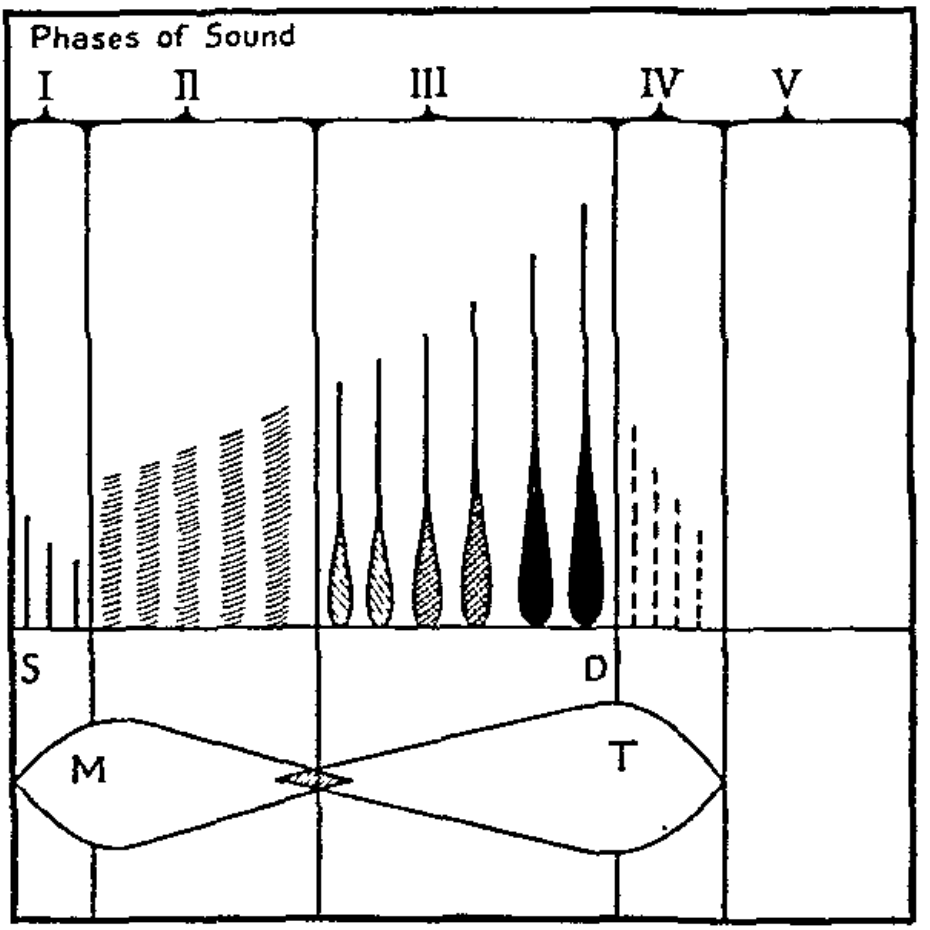


FIG. 2.—The five phases of sound of the auditory sequence. I, clicks; II, murmurs; III, thuds; IV, dull sounds, V, silence. M, zone of murmurs, caused by eddies in the blood current; T, zone of thuds, caused by vibrations of the arterial wall. S, level of systolic pressure; D, level of diastolic pressure.

1. The First Point.—The first point coincides with the appearance of the first sound or click. In reality, it measures the sum of the systolic pressure head, velocity head and such increment as may be due to water hammer.[10]

These clicks together constitute the first phase, which is usually only of a few millimetres' duration.

2. The Second Point.—The second point ushers in the second phase, and is recognisable by either the addition of a soft blowing murmur to the clear sound, or its entire replacement by the murmur. This phase is usually longer than the first one.

3. The Third Point.—The third point occurs when the murmur disappears and gives place to a zone of sounds somewhat similar to those of the first phase but louder, and becoming gradually more accentuated and throbbing in character. This crescendo sequence of clear thudding sounds constitutes the third phase of sound.

4. The Fourth Point.—The fourth point is reached at the instant when the terminal loudest thuds of the third phase abruptly lose their special quality and are succeeded by a zone, either short or long, of muffled and dull sounds, tailing off into silence. These weaker sounds represent the fourth phase, the *beginning* of which, *i.e.*, the first dull sound following the last thud, is the auditory index of the minimal (diastolic) pressure.

5. The Fifth Point.—The fifth point registers the disappearance of all sound.

Analysis of the Five Phases of Sound

Experimental and clinical researches on the production of the five phases of sound have been made by numerous workers,[10, 11, 12, 13, 14] in particular by Gallavardin [15, 16] and his pupil Barbier,[17] to whose writings the reader is referred for fuller details.

These observers have established the presence of *two zones* in the curve of sound: *one* occupying the upper half of the curve with its maximum near S, *caused by eddies in the* **blood current** partaking ordinarily of the character of murmurs; *another* occupying the lower half of the curve with its maximum near D, *composed of sounds originating in the* **vessel wall**, and whose intensity is clearly related to the state of excitability of the periarterial sympathetic system. Normally these two zones overlap in the middle

of the auditory curve which is the synthesis of these two zones of differing origin (Fig. 2).

Detailed investigation of each sound phase [17] reveals the following characteristics (Fig. 3) :—

First Phase.—The sounds are small and light. Frequently the first click is clearer and sharper than the few which immediately follow it. Very often the sounds speedily take on a murmuring tone, which relegates them to the second phase, the first phase then being much abbreviated.

The sounds are light because it is only the crest of the systolic wave which causes the walls of the artery slightly to separate with the least amount of vibration. The duration of opening of the artery is very brief, and the amount of blood in movement minimal, both of which conditions are unfavourable for the production of any considerable blood murmur. If the first click is a little stronger than the following ones, the reason is because it has to open up the obliterated artery, while succeeding waves have not to overcome a like inertia, since they come upon an arterial door which does not so readily shut.

The first sharp click, which denotes the beginning of the first phase, is the auditory index of the maximal (systolic) pressure.

Second Phase.—This zone of murmurs, with its phenomena more defined and easier to follow, is due to eddies in the blood current as it traverses the arterial constriction and drops from a raised pressure to a lower one on entering the uncompressed portion of the brachial below the armlet. Thus the murmurs of the second phase are related with a lack of pressure balance between the segment of the artery above the armlet and the segment distal to it, which is in a state of relative hypotension.

That these murmurs really originate in the blood is proved by the following observations : (*a*) An Esmarch's bandage applied to the middle third of the forearm induces an increase of pressure at the level of the distal arterial segment, and so has the effect of suppressing the murmurs.[10, 11, 12] (*b*) The murmur phenomenon is much more obvious with gradual decompression than with a rising external pressure. In the former, the first blood waves find the artery almost

empty, hence the murmurs; in the latter, the progressive compression exercised by the armlet has from the outset hindered the escape of venous blood, whilst the arteries remain full; the arterial segment distal to the armlet is embarrassed, its pressure is kept high, and murmurs are, therefore, absent. (*c*) The most important proof, however,

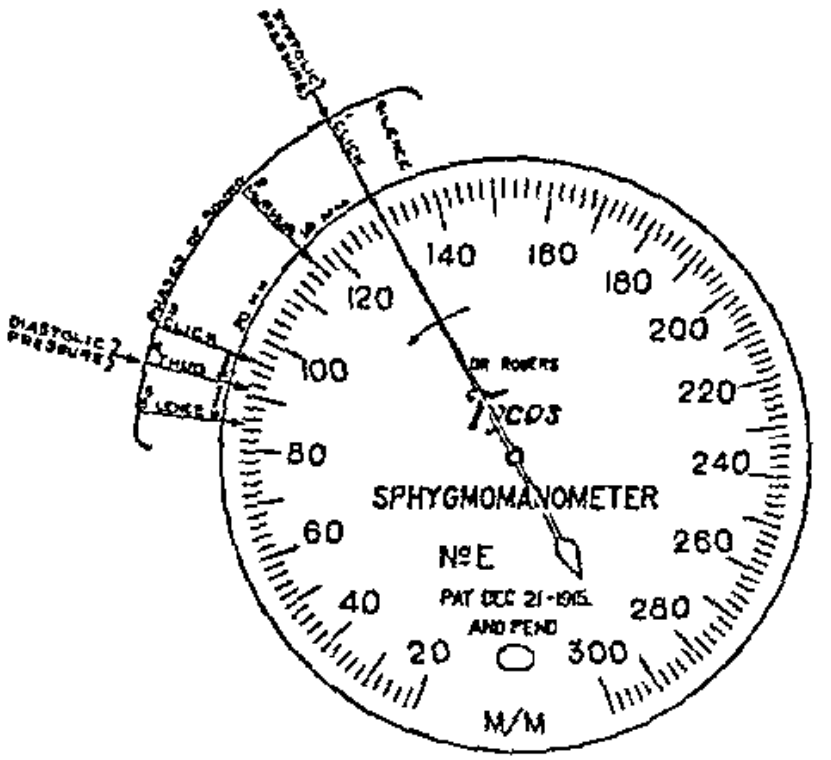


FIG. 3.—Diagram to illustrate the characteristic change of tone and approximate length of the phases of sound heard through the stethoscope.

1. The first sound A sharp click The index of systolic pressure. In the above diagram this phase covers 14 mm.

2. A zone of murmurs resembling heart murmurs Is pictured here as lasting 20 mm.

3. A zone of clear thuds somewhat like the first sound, but generally louder, and continuing for about 4 mm. of the scale reading.

4 Dull sounds. The beginning of this fourth phase is the index of diastolic pressure. Of variable duration from 3 mm even up to 55 mm

lies in the fact that the murmur is a transitory phenomenon. After prolonged compression for twenty to thirty beats, the murmur lessens and then disappears, the armlet acting like an Esmarch's tourniquet in impeding venous outflow.[17] (*d*) The murmur can be brought back by promoting evacuation of the distal segment by light massage of the superficial

veins of the forearm or by movements of the hand. (*e*) The murmur zone corresponds to a period during which oscillometry registers only feeble expansions of the vessel wall.

Arterial tonus, nevertheless, can come into play even though the phenomenon be related to the blood much more than to the vessel wall.

Third Phase.—The sounds of the third phase assume more or less quickly a special quality. They become loud and intense, sounding "like the throbbing of a drum or the beat of a gong,"[16] and, after reaching a maximum, abruptly lose their special character. It is interesting to note that this series of thuds coincides exactly with the phase of brachial vibration, whilst the oscillometer needle during this period also manifests a definitely abrupt swing (p. 47).

During this phase the artery does not undergo complete collapse, but, opened by each larger systolic wave, resembles a door closed by a gradually relaxing spring and opening with a sudden gust of wind. Compression of the tissues caused by the armlet represents the spring closure, whilst the systolic wave represents the sudden gust of wind. The sounds produced in the wall of the artery as a result of this brusque opening are directly proportional to the forces of opening and closing. If the artery, feebly closed by an insufficient force, is violently opened by a strong wave, a loud vibrant tone is produced. The variable resistance against which the door closes is the armlet, which, adapted to the artery, with each expansion sustains an impact.[17] "A loud tone may be produced by a stiff artery and a slow stream, or by an elastic artery and a rapid stream."[18]

"The third sound phase is thus directly related to the tonus of the artery under investigation; normal or increased tonus will give for the same blood wave an arterial sound vibration much more thudding and loud than will an artery with flaccid walls, and it is for arteries with normal or heightened tonus that the simile of the tap of the drum can be employed with greatest accuracy. The sympathetic intervenes as the tightener of the arterial drum, regulating at each instant the sonorous properties of its wall."[17] This sympathetic action, evoked by the constriction of the armlet,

varies considerably in different persons, even under like conditions of blood pressure and heart rate. The proofs of this are that (1) normally the arterial sounds are more intense at the first than at subsequent readings. With repeated compression they diminish from minute to minute; the sympathetic, which at the outset has put forth its maximum action, flags, and the artery little by little loses its sonorous properties. (2) Rapid compression up to 300 mm. always induces a sudden increase in sound, which is transitory and followed by rapid diminution. Strong compression provokes energetic vaso-constriction, but the sympathetic tires more quickly in response to a single violent effort. (3) Application of local cold to the artery, as by a compress soaked in ether introduced between the arm and armlet, brings about marked reinforcement of the sounds,[19] for cold acts as a potent vaso-constrictor. (4) Injections of adrenalin or pituitrin cause also a temporary reinforcement. (5) Excess or lack of sympathetic tone finds in the arterial sounds a faithful mirror which renders the auditory method a simple means of investigating sympathetic action.

In irregular hearts there is marked difference in intensity between successive sounds ("tonal arrhythmia" of Goodman and Howell).[20] Loud sounds occur with rapid blood flow; and it is interesting to note that in anæmic states the sounds are also loud and clear, although here they bear no true relation to the cardiac energy. This paradox is due to loss of vasomotor tone in association with atrophy and lack of nutrition of the muscular coats. "In polycythæmia the sounds have a curious, dull, sticky character, and cannot be differentiated into phases. In not all cases can all phases be made out. It is usually the fourth phase which fails to be heard. In such cases the loud third tone almost immediately passes to the fifth phase."[18]

Fourth Phase.—*The change from loud thuds to dull sounds, which marks the beginning of the fourth phase, is the auditory index of the minimal (diastolic) pressure.* "It is the appearance of dull sounds and not the disappearance of all sound that indicates the diastolic pressure."[18]

The sounds are inconstant, dull, weak and sometimes barely perceptible, being proportional to the suddenness and

amplitude of the arterial oscillations. This phase is due to change from an external pressure sufficient to cause distortion of the circular tube to one insufficient to cause any flattening, *i.e.*, an external pressure equal to the internal diastolic pressure.

The duration of the fourth phase varies from 3 mm. even up to 55 mm. Hg, a short phase being usually associated with high pressures, whilst a long phase is often met with in normal or low pressures. The average length is much greater than formerly believed, and there is no constant relation to variations in pulse rate, systolic pressure or differential pressure.[21]

Fifth Phase.—The fifth phase of silence ensues when the artery is relieved from the compression exercised by the armlet.

Although the auditory series of sounds follows a broad general type, shown in Fig. 5, denoting good functional equilibrium between heart and arteries, yet notable individual variations may occur.

Anomalous Auditory Curves

The physiological theory advanced by Barbier[17] of a "mixed" blood and vessel wall origin of the sounds of the auditory curve explains the frequent variations and gives to each form of curve a diagnostic value.

I. **Variations in Intensity of Sounds produced in the Arterial Wall.**—A long and loud third phase betokens vigorous cardiac systole or a moderate degree of arteriosclerosis, but if the latter be of high grade or attended with calcification, the intensity of the sounds is much diminished. The sympathetic plays an important part also in arterial tonality, and specially in the genesis of vibration, which strengthens with heightened sympathetic excitability (hypersympatheticotonus), as in larval or developed Graves' disease, and weakens (*a*) in diminished sympathetic excitability (hyposympathetic tonus), after stimulation by cold or adrenalin; (*b*) in all valvular or circulatory insufficiencies, including chronic heart-muscle weakness; and (*c*) in arterial hypotonia.

Abnormal Persistence of Sound Production.—There may be persistence of sound through the normally silent fifth phase.

(1) In cases of collapsing pulse, even in the absence of compression by an armlet, sound vibrations are set up in the arterial wall. Their production depends upon reduction of diastolic distension combined with a considerable or sudden systolic increase of pressure head. Hence the loud "pistol-shot" sounds of aortic insufficiency in the presence of hyperpiesis, caused by vibrations resulting from sudden distension of the arterial wall—a water-hammer effect.

(2) With increase in sympathetic excitability causing arterial constriction, as in hyperthyroidism, and in slight arterial hypertonus.

(3) Sometimes, in the absence of disease, distinct, though weak, thuds may persist far below the diastolic level. Under certain conditions there may be persistence of sound after the cuff pressure has fallen below the pressure necessary to cause any flattening of the artery, but is still sufficient to diminish the arterial calibre from diastolic size as compared with the artery proximal and distal to the length of tube compressed.[10]

II. Variations in Intensity of Sounds produced in the Blood Current.—Prolongation of the second phase of murmurs caused by the blood current is due to (*a*) anæmic states, which constitute the chief cause. Sometimes, in extreme cases, a true thrill, accompanying the murmur, may be felt by the finger placed over the brachial artery, which thrill disappears along with the vibration at diastolic level. Exaggeration of the murmur zone may also be due to (*b*) sympathetic influence producing a localised arterial hypertonus; (*c*) modification of the arterial pulse of the nature of anacrotism. The murmurs may be entirely absent in hyperpiesis or aortic stenosis, the rate of arterial blood flow not being rapid enough to cause a murmur during the second phase. An *auditory gap* results, since there is nothing to supplement or replace the arterial sound which during that phase weakens or disappears.

In general terms it may be stated that modifications affecting the whole of the auditory curve are almost always

due to a central cause, cardiac or aortic, while modifications of a portion only of the curve are usually of peripheral origin, arterial in kind. Further, the auditory curve may be deficient at one or other extremity, or in the middle may present a complete gap, no sound being audible over a range usually of about 25 mm. in the position where one would normally expect the second phase. Instead of a gap only faint sounds may be appreciable.

2. The Tactile or Palpatory Method

In determining systolic pressure according to the principle of Riva-Rocci the usual manner of applying the tactile

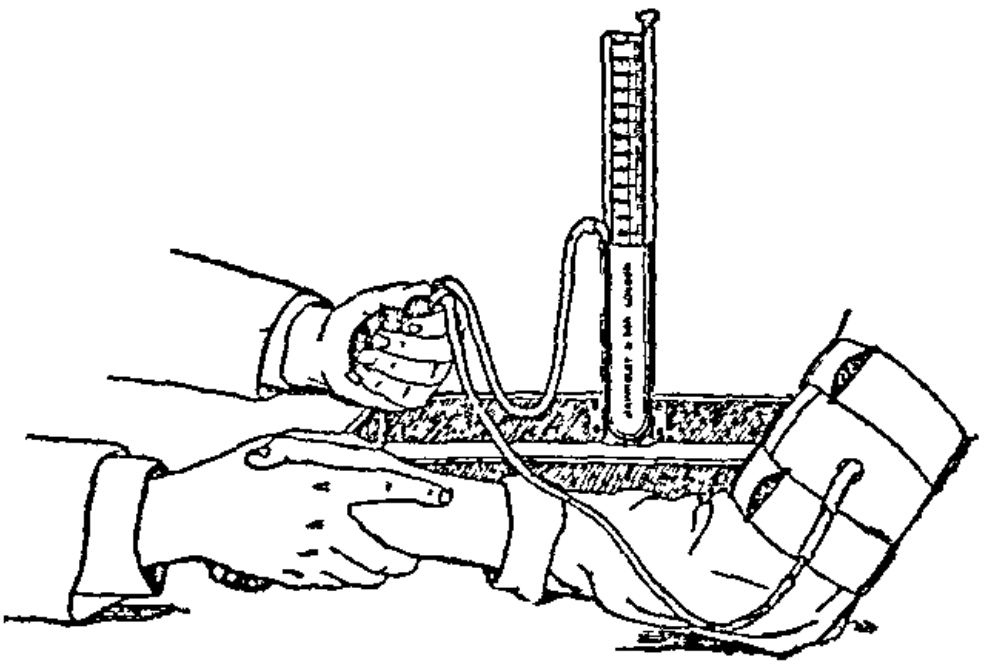


FIG. 4.—The tactile method of estimating arterial pressure by palpation of the radial artery

or palpatory method is by means of the pressure bag gradually to compress one artery—the brachial in the arm—while noting the disappearance of the pulse by palpation over a totally different artery—the radial in the forearm. The pulse disappears at the moment when the blood stream no longer possesses enough energy to overcome the circular resistance opposed to it at the level of the compressing armlet, *i.e.*, when the pressure within the armlet is equal to the maximal arterial pressure at the given point. *On*

progressive decompression, appearance of the pulse is the tactile index of the maximal pressure. In order to avoid extra error the only *correct* way of radial palpation is to allow the patient's forearm to rest in a position midway between pronation and supination while the observer lightly grasps the patient's hand, at the same time palpating the radial artery with the extended index finger, as shown in Fig. 4.

Since the systolic pressure is gradually falling and the diastolic pressure is gradually rising between the brachial artery and the periphery, as confirmed by numerous comparative experiments which I have performed with different types of arterial pressure instrument,[22] the tactile method, *as usually practised*, has never seemed to me to give *exact* indications of either maximal or minimal pressures, and thus, for practical purposes, in the main has rightly been superseded by the auditory method.

Notable differences, which are not, however, necessarily constant, are at times found between arm and forearm readings (as also between the two arms, or between arm and leg readings). Such are apparently caused by local conditions of unequal contraction or hypermyotrophy. "Diminution in the calibre of an artery, if extreme, may influence the propagation of the systolic wave, but under the ordinary conditions of blood-pressure estimation in the arm this factor is a negligible one "[23]

Apart from local influences due to the state of the arterial wall, systolic pressure cannot be higher in the forearm than in the arm, and although there is no evidence of any *important* loss in transmission of the pulse wave from brachial to radial, since normal arteries, even under widely differing conditions of tone, possess good power of conductance of the systolic wave, palpation of the radial artery usually gives a reading *from about 2 to 10 mm. lower than the brachial systolic pressure, which is the pressure that in reality we wish to determine.*

I have laid stress upon this point because Barbier[17] believes that because of the anomalous auditory curves, which are apt to present difficulties, it is indispensable to supplement the information obtained by the auditory

method as to the exact situation of the systolic pressure by radial palpation, whilst MacWilliam and Melvin [6] use it as a check in the routine application of the auditory method. They state that the auditory reading should always be *at least as high* as the tactile (radial) reading, though very often it is somewhat higher. An auditory reading lower than the tactile one shows that the conditions are such as to invalidate the accuracy of both systolic and diastolic auditory indices, a result which is often due to faulty application of the tambour over the artery, anatomical variation of the latter, etc., and may be remediable.

Whenever possible I prefer a more exact way of assessing the normal relation between auditory and tactile systolic and diastolic indices by placing the finger below the bell of the stethoscope or auditory tambour and palpating the *brachial* artery, when *the first pulsation will be found closely to correspond with the first audible click*, which denotes the level of the maximal pressure, whilst the last notable vibration coincides with the last loud thud, which marks the level of the minimal pressure. Unfortunately, however, the brachial artery usually lies too deeply or is too embedded in fat for this procedure to be applicable, in which events recourse must perforce be had to radial palpation, which, although not as accurate, gives approximate and comparable readings. An addition of 5 mm to the reading obtained by palpation of the radial artery will give a fairly accurate result.

With an intelligent patient it is often possible to check readings by the patient's own sensations. At the moment of the first click, a slight sensation may be felt beneath the armlet as well as an alteration in intensity of the throb at the beginning of the fourth phase.

Cases of nervous disorder attended by extreme tremor present difficulties in estimation by means of auditory and tactile methods, and it is not always possible to do more than approximate to an exact reading. Light hypnosis has been suggested, but possesses the disadvantage of cutting out the psychic factor, the degree of which at the onset it may be useful to determine, although for precise estimation, elimination of the psychic factor is desirable, and may largely be attained by determination of the *residual* pressure (p. 27).

3. The Vibratory Method

This method is really a modification for a special purpose of the tactile one, and is employed chiefly for the purpose of determining the diastolic pressure by palpating the brachial artery just below the armlet during decompression, when the finger perceives at first simple beats increasing in

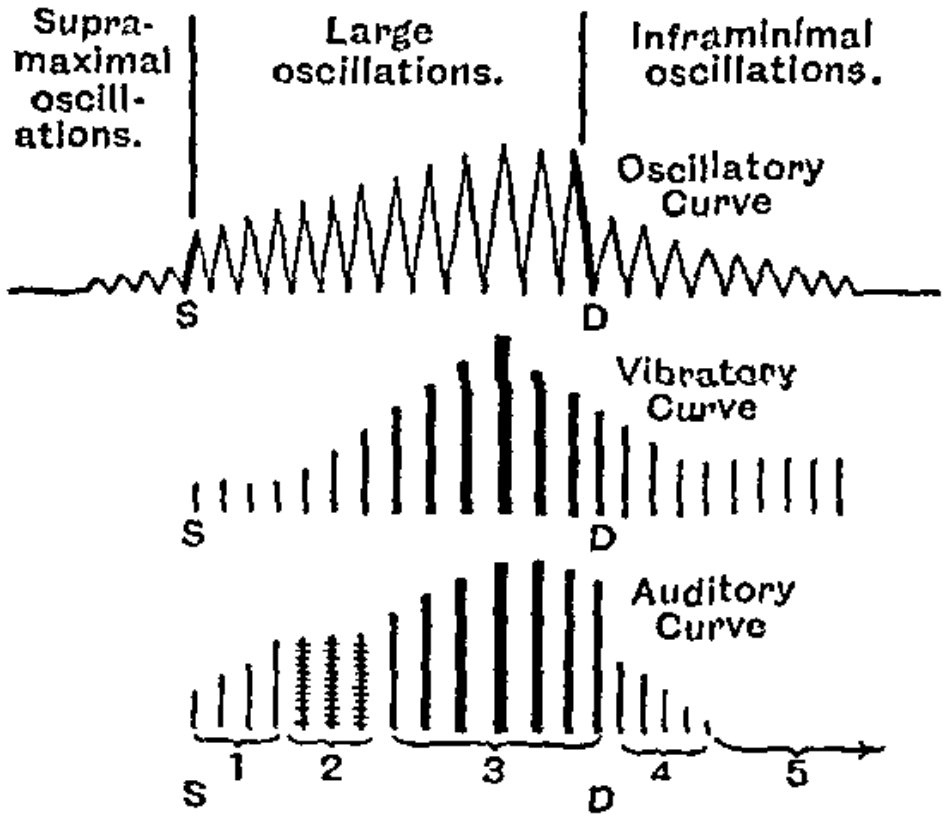


FIG 5.—Diagrammatic comparison of curves obtained by the use of the oscillatory, palpato-vibratory and auditory methods of estimating arterial pressure Curves to be read from left to right, showing vertical correspondences between the levels of S, the systolic pressure, and of D, the diastolic pressure. The figures below the auditory curve refer to the five phases of sound: 1 = clicks, 2 = murmurs, 3 = thuds, 4 = dull sounds, 5 = no sound In comparison with Fig. 2, the third phase shows slight lessening in intensity of sounds towards the end, but the fourth point is well defined.

intensity shortly followed by a series of vibrations which, after attaining a maximum, decrease and then vanish. *This point of transition from vibration to no vibration marks* the change from the third to the fourth phase of sound and has been strongly advocated by Gallavardin [16] as a reliable means of estimating with accuracy *the diastolic index.* It

can also be employed as an additional check on the auditory method, especially in difficult or doubtful cases, or if the observer's hearing be not good. Observations in my wards showed that the diastolic pressure could readily be determined by this method in about 60 per cent. of cases where the ear was unable to gauge the point at which loud thuds gave place to dull sounds.

4. The Oscillatory Method

The oscillatory method is founded upon the original observation of Marey [24] (1876) upon the amplitude of pulsations of a segment of a limb subjected to decreasing circular compression. It is only satisfactory with clinical instruments of visual type possessing a large dial and a long delicate registering needle, such as the Tycos "Clinic Pattern" (which, however, does not register higher readings than 300 mm. Hg), or preferably with Boulitte's Universal Oscillometer, which operates with maximal sensitiveness, and is thus one of the most accurate aneroid instruments yet available. The oscillations of the little needle in the ordinary Tycos aneroid and other instruments of similar type are so tiny that it is difficult to differentiate the systolic oscillation from those which precede and follow it.

5. The Graphic Method

By the systematic use of this method many additional data of fresh value and significance have been afforded. In comparison with the foregoing, this latest advance possesses three important advantages in that it yields (1) automatic, (2) graphic and (3) permanent records of arterial pressures, as well as of individual pulse characters.

Apart from laboratory instruments of older date, such as those of Gibson or Erlanger (*vide* Chapter XV.), various types of clinical instrument have recently been evolved, namely the Tonoscillograph of Plesch, the Arterial Oscillograph and the Portable Registering Oscillometer of Boulitte, and the Tycos Sphygmotonograph.

Conclusions

Parallel in principle, the five methods of estimating arterial pressure are akin in their results.[17, 25, 26, 27] For the large majority of cases—about 90 per cent.—the first four methods can readily be employed, even by a novice. In the remaining 10 per cent. sometimes they are difficult, and at other times, though rarely, impossible.

Each method naturally has its strong advocates, but in the cases which are really doubtful seldom is any one method found to possess distinct advantages over the others.

As a rule the auditory method suffices, but although it is the most convenient and accurate yet discovered, nevertheless when used by itself to determine the systolic pressure it has some limitations which militate against its universal application.

The best procedure is that which is liable to the fewest errors, and since unqualified adhesion to the auditory method may mislead, I am in agreement in recommending for unusual cases **the employment of a combined auditory-tactile method,** which presents no added difficulties and possesses many and great advantages.

The True Index of Diastolic Pressure

Uncertainty has been felt in regard to the essential question as to what point in the series of auditory phenomena is to be taken as a true indication of diastolic or minimum pressure

Korotkow [8] gave as his criterion the "end tone," *i.e.*, change from loud to dull sounds (Figs. 2, 3, and 5), which occurs at the beginning of the fourth sound-phase. Certain of the earlier observers who followed him regarded nevertheless the lower limit or extinction of sound (fifth phase) as the index, since it has been stated to call for the simplest technique, and to be more easily determined by unskilled observers than any other point. Many others again have considered that the true diastolic point must be somewhat higher, and that it should be placed just where the clear, loud note becomes suddenly dull and muffled or distant.

As the result of the investigations of Lang and Manswetowa,[28] Fischer,[29] Warfield,[18] Erlanger,[30] Taussig and Cook,[31] Weysse and Lutz,[32] supported by Oliver [33] in the third edition of his book, and more recently by Gallavardin,[16] any doubt in accepting the beginning of the fourth phase as the index of diastolic pressure has been swept away. The admirable and exhaustive critical studies of Professor MacWilliam, Spencer Melvin and Murray have further succeeded in establishing the correctness of this criterion on a firm and unassailable basis. From experimental observations (1) with a circulatory schema,[5] (2) with animals of sufficient size, such as the sheep,[7] and (3) on man,[2, 21] using concurrently the auditory, the tactile and the graphic method with Erlanger's apparatus, and comparing these with visual results obtained by employing the Pachon sphygmo-oscillometer with the brachial armlet, they find that "weakening and dulling coincides with the point at which the arterial tube just ceases to be flattened by the external pressure between the pulse-beats; . . . vibration associated with the sudden change in the shape of the tube is evidently responsible for the character of the sound," and that this point proves "a very accurate guide to the intra-arterial diastolic pressure as shown by the minimum manometer." They are satisfied that all the evidence as to diastolic pressure gained by other methods, and much more also, can be better obtained by the quick and simple auditory method.[6] Later the substantial correctness of systolic and diastolic criteria was verified by Erlanger [30] in a series of photographic records of movements of points on the exposed artery of the dog whilst undergoing decompression similar to that in clinical arterial pressure determination in man. He stated that the beginning of the first sound phase develops shortly after the compressing pressure falls below the systolic point, and that the change from sounds of sharp quality to dull sounds fairly accurately indicates the moment at which the compressing pressure falls below the diastolic.

Notwithstanding this volume of conclusive evidence (to which the reader in search of fuller information is referred) as to what constitutes the true criterion of diastolic pressure, in

the interests of statistical uniformity it is regrettable to find that certain workers, knowing the exact index, deliberately prefer the much less exact. Notably is this the case with certain American life assurance offices, all of whose medical examiners have been trained to take the diastolic pressure at the very end of the fourth phase, just before the beginning of silence, for the alleged reason that this point calls for the simplest technique, and is more easily determined than the end of the third phase.[34, 35] That such procedure is incorrect is amply proved by Melvin and Murray,[36] who have demonstrated that *the length of the fourth phase is subject to very considerable variation. It may be only a few millimetres, or may extend to 55 mm.* In the latter case, as they state, utterly fallacious results would be obtained by taking the lower limit of the sound as the diastolic index.

Personally I think that objections on the ground of expediency to acknowledging the beginning of the fourth phase as the true index of diastolic pressure are more fanciful than real, for, given average auditory acuity and reasonable care and attention, no difficulty is experienced in noting the exact transition from loud to dull sounds in a normal auditory curve. Practically the only cases which are likely to mislead the unwary are those in which the third phase is weak or absent as a result of insufficient blood flow through the brachial either from local congestion distal to the armlet or from loss of cardiac strength, and in aortic regurgitation, where the sound is often continued over the fifth phase. Often, too, in aortic regurgitation the familiar loud systolic thud and less frequently the double murmur of Duroziez are audible over the arteries not subjected to compression. The presence of such sound does not, however, as a rule prevent recognition of the diastolic index, the sudden weakening and dulling being usually very well marked. In such cases the tactile systolic index should be employed simultaneously as a check.

audible sound (the click) exactly corresponds to the first brachial pulsation. In this way we are enabled definitely to establish the first sound perceived as the criterion of systolic pressure. Failing this, there is a correspondence nearly, although not quite, as exact between occurrence of the first sound and of pulsation in the radial artery, the return of the pulse wave in the latter being from a few millimetres up to 10 mm. or so later than the appearance of the first sound. Hence the tactile method constitutes a useful check upon the accuracy of the auditory method.

TECHNIQUE OF ARTERIAL PRESSURE ESTIMATION BY THE AUDITORY METHOD

For any reading of arterial pressure to be accurate and trustworthy it is essential that the subject should be at rest, which implies both cessation from physical motion and from psychical disturbance. Such desiderata are best attained by not attempting to measure the blood pressure until sufficient time has elapsed for the patient to have calmed down from any excitement or apprehension incidental to the medical examination and to feel perfectly at ease.

At the first consultation it will be found most convenient to take the observation after the history but before the remainder of the clinical examination.

The following constitutes a description of the auditory method of estimating arterial pressure (vide *Fig.* 1), *which I recommend as the quickest, simplest and most accurate yet devised. It is applicable to any modern type of instrument :—*

1. The armlet should be smoothly and evenly adjusted as high up the arm as possible, the upper margin touching the axillary fold. The lower margin is thus brought well above the bend of the elbow. Care should be taken that the middle of the bag is towards the inner side of the arm, so as to ensure effective compression of the brachial artery. The armlet must not be applied too tightly. This matter is easily arranged with the older form of stiff outer cuff; with the newer soft bandage-like armlet the folds should gradually be brought down so as to enwrap the whole

width of the pressure bag, and the last 2 inches of the tail neatly tucked under the preceding turn.

2. The patient's arm, which is bared, should be allowed comfortably to lie, with all its muscles completely relaxed, upon a support of such height that the armlet is brought to the same level as that of the heart.

3. At this juncture it is well to distract the patient's attention by determining with the finger the rate and characteristics of the pulse, since both arterial pressures and pulse rate should be taken under the same conditions.

4. *Now explain to the patient that the band round the* arm will tighten for a minute or so, but that this temporary pressure is quite harmless Apprehensiveness on the part of a sensitive patient may be further allayed by slight inflation of the bag for a few seconds in order to accustom him to the novel sensation produced by compression of the limb.

5. Rapidly inflate the air-bag of the armlet to a pressure of about 110 mm Hg

6. Adjust the bell of a stethoscope, or an auditory tambour with rubber band for keeping it in place, below the armlet and over the brachial artery just above the bend of the elbow to the inner side of the biceps tendon, when in the majority of all cases, whether in health or disease, at or about a pressure of 110 mm. Hg, successive clear and loud thudding sounds will be audible as the blood passes through that portion of the artery which is partially constricted by the armlet.*

7. Quickly raise the pressure still further until all sound disappears, and the brachial and radial pulses are completely extinguished.

8. Slowly open the release valve, thus gradually lowering the external air pressure, and, according to the kind of instrument in use, note accurately either the height of the mercury column or the figure reached by the hand on the dial at which the first definite click is heard. **The first audible click on decompression following obliteration indicates the systolic pressure.** This point is a few millimetres

* A practical way of determining whether the tambour is in the correct position is to apply local increase of pressure, when, if the tambour is directly over the brachial artery, the radial pulse will be felt to lessen or disappear.

higher than the return of the pulse to the finger placed over the radial artery at the wrist.

9. As the pressure continues gently to fall, the clear, sharp click gives place to a soft murmur of variable duration, which is succeeded by a longer phase of clear, vibrant and sonorous thuds, which soon reach a maximum, finally becoming dull and muffled before total disappearance. **The point at which transformation of the clear, loud thud into a dull sound takes place indicates the diastolic pressure.** This change is usually sudden or rapid, but occasionally may be more gradual.

10. The mercury in the manometer is now allowed to return to zero by completely emptying the pressure bag of air and leaving it so for a few moments in order to permit the venous stasis to disappear and the circulation in the upper limb below the armlet to return to normal.

11. Discard the result of the first estimation, which should always be regarded as a rough approximation, and concentrate closely on the next two or even three readings, in order to determine the basic or residual pressure of the patient at the time of estimation by eliminating, through compression of the brachial, any element of hypertonia that may be present. Readings subsequent to the first are very often lower. Especially is this true of the systolic pressure, for the reasons given on pp. 29–32. Each complete estimation should not take longer than one minute. Continue until a constant level is reached, *the residual arterial pressure.* If there is no drop in pressure within three minutes, and the high readings are thought to be accidental, without telling the patient that the pressure appears too high, get him to return and again estimate the pressure levels on a second visit.

12. *The residual pressure, which is what one desires to record, is the lowest constant pressure reading.*

This should forthwith be entered in the notes of the case according to the author's formula, which constitutes

The Complete Arterial Pressure Picture (Fig 6).

The complete arterial pressure picture includes :—

(1) The figure for the maximal pressure.

(2) The figure for the minimal pressure.

(3) The figure for the differential (pulse) pressure.
(4) The rate and characters of the pulse.
(5) The figure obtained by multiplying the differential pressure by the pulse rate.

A simple and compact formula is thus obtained, which can be expressed with rapidity and accuracy as

$$\frac{S.}{D.}\ \text{D.P.} : \text{pulse rate and characters} : \text{D.P.} \times \text{P.R.}$$

e.g., $\frac{165}{100}$ 65 : 78, small, irregular, wall thickened and tortuous : 5070.

In grouping large numbers of cases for statistical purposes into high, medium and low pressure classes, the graphic method of representation shown in Fig. 6 has been found to be of practical utility.

All comparative observations should be carefully made in a comfortably warmed and silent room, with the patient's limb in the same relative position of rest at the level of the heart, *as far as possible at the same time of day*, preferably midway between meals, *on the same limb and with the same type of instrument, all psychical disturbance being brought to the irreducible minimum.* The first observation is apt to be higher than the subsequent ones, and should always be corrected by others taken before the end of the first interview, when it will usually be found that the later readings tally very closely. The author's practice is to take at least three readings, and to record in the case-notes the results of the third or subsequent constant reading which denotes the required residual pressure.

I have seen so many erroneous deductions drawn through failure to comply with these few simple precautions that it cannot be too strongly urged upon every observer that reliable comparisons can only be instituted between blood pressure tests in the same and in different individuals when such are made under similar conditions. If the type of instrument be changed, this fact should always be noted, since all the older forms with narrow armlet, etc., give readings which are far too high.

Although the question cannot be regarded as definitely

settled, the consensus of opinion points to maximal and minimal pressures being a few millimetres higher in the standing than in the sitting position, and in the sitting position a few millimetres higher than when lying down.

Fallacies in Estimation of Arterial Pressure by the Auditory Method

A. **Sources of Error affecting both Systolic and Diastolic Pressures.**—1. Serious error may be introduced by unnecessarily prolonged compression during estimation, thus causing stasis of blood in the limb

2. Error may be caused by the bell of the stethoscope or the auditory tambour not being placed directly over the artery, or by the bell being tilted at one edge, or by pressure upon it being excessive.

3 Error may be caused by the existence of an artery of small and insufficient size.

Unavoidable difficulties through any of the above causes should be of extreme rarity. If the chance of error be suspected, a combination of the tactile with the auditory method serves as a useful check in the determination of the correct indices.

The results which follow are entirely concerned with the estimation of systolic pressure, since *the diastolic reading is little, if at all, influenced by the chief sources of fallacy* regarding local conditions of altered conduction, resistance of arterial wall, or reflections from the periphery, *which may affect the systolic reading.*

B. **Sources of Error causing Over-estimation of the Systolic Pressure.**—These depend upon—

(*a*) *Factors External to the Artery under Compression.*

1. Considerable adiposity of the limb.

2. Œdema of the limb. These conditions are infrequent, and invariably prevent accurate readings.

3. Cyanosis of the limb. For general cyanosis no immediate remedy is applicable. Cyanosis due to local congestion induced by too lengthy compression of the armlet can be obviated by (*a*) rapid filling of the bag with air ; (*β*) taking care that decompression is not too prolonged ;

(γ) allowing sufficient time between each reading for any cyanosis to disappear.

4. Active contraction, tonic or clonic spasmodic states, such as cramp or tetany of the muscles of the limb. This source of error can be avoided by never taking the blood pressure until and unless the limb is completely passive with its muscles at rest. In certain disorders of the nervous system, however complete muscular relaxation is impossible, when perhaps one of the other limbs may not be affected and can be utilised for purposes of estimation.

(*b*) *Factors due to Variations in Resistance of the Arterial Wall*

1 **Transitory Arterial Hypertonic Spasm** may be due to excitement and apprehension consequent upon the unaccustomed constriction of the armlet and painful feelings of swelling of the limb These emotional effects are manifested by quickened pulse rate, and can be removed by calming and reassuring the patient as well as by shortening the time of estimation

2 **Continued Hypertonic Contraction of the Arterial Muscle** definitely influences compressibility, but in the brachial artery a degree of contraction sufficient to cause an error of more than 30 mm Hg is improbable in adults, and is usually considerably less than this, whilst in children it is negligible

In common with other observers, W Russell [37] has laid great stress on the state of the media, and has repeatedly pointed out the effect of arterial contraction on clinical readings He believes that arterial hypertonus rather than hyperpiesis is mainly what is measured by the obliteration method The results of his experimental work with rubber tubes and with arteries treated with formalin, etc., are, nevertheless, inadmissible in the case of living arteries, since the conditions are too dissimilar for comparison. Neither can his observations with vaso-dilators (erythrol tetranitrate, etc) be accepted, since he appears to ascribe the reduction of the readings to relaxation of the wall of the brachial artery whilst ignoring the effect in lowering of systolic pressure due to opening up of the peripheral vessels by the drug.

3. Calcification of the Arterial Wall.—The common idea of calcification, wherever in the body it occurs, is that the process results in a condition of stony hardness. But MacCordick [38] has proved that *during life calcified arteries are not rigid.* At operation and at autopsies immediately following death such arteries are found to be pliable and to cut readily.

In the "pipe-stem" radial, for example, the calcareous matter is deposited, not in the intima, but in the media in a state which is identical with that of unset mortar. Mortar sets by conversion of calcium hydroxide into calcium carbonate by absorption of carbon dioxide from the air. Kept in alkaline media, or in bulk away from the air, mortar remains unset, but sets when placed in an acid medium. Identical changes take place within the body. During life the reaction of the body tissues and fluids is alkaline, but becomes acid shortly after death, and the gas which charges the blood within the arteries after death is mainly carbon dioxide. Hence "atheromatous ulceration and saucer-like plaques with rupture of the surrounding endothelium are *post-mortem* products. Calcified areas in the intima become set and rigid before muscular contraction of the aorta gives way, and the tube undergoes dilatation. The result is that tension upon the endothelium at the edges of the plaque induces rupture of the lining."

It is to the valuable work of Professor MacWilliam,[39] E. I. Kesson [4] and G. Spencer Melvin [2] that we are indebted for the most exact and useful knowledge we possess both as to verification of the criterion of diastolic pressure and influence of the arterial wall on blood pressure measurement. These observers, working on broader lines than their predecessors, have undertaken a series of clinical studies in the light of careful experimental data derived from investigation of excised living arteries in different conditions of contraction and relaxation, and of abnormal and thickened arteries as well as normal ones. From their clinical study they draw the following conclusions :—

"Estimations of systolic blood pressure by the obliteration method, when made with suitable precautions, give substantially correct results in ordinary conditions of normal

health and also in the great majority of cases of illness. Even when the disease affects the vascular system with thickened arteries, etc., the indications are in the majority of cases approximately correct, the readings ranging from moderate or low to very high values. It is only in a minority of cases that serious error is liable to occur, in the direction of over-estimation.

"In some such cases the influences of local conditions may be very important, especially the presence of abnormal resistance in the arterial wall, depending mainly at least on contraction of the muscular coat In such conditions very different readings may be obtained from the same person on the same occasion according to the limbs or parts of limbs examined, the using of first or later readings, etc.

"Continued or repeated compression, with comparison of the two sides, etc , affords a valuable method of detecting the presence of such error, though not invariably decisive; in some instances considerable disturbances of blood pressure may occur Results, both positive and negative, obtained by this method show a striking parallelism to those yielded by excised surviving arteries" [6]

Thickening of the arterial wall may be held to include atheroma, sclerosis of intima, and often of adventitia, or increase of media—the hypermyotrophy described by Russell,[37] Savill,[40] Dickenson and Rolleston[41] and others

Upon compressibility arterial thickening has no appreciable effect, and calcification only a slight effect (p. 31); it is contraction that tells Hence the whole matter may be summed up by the statement that *in the vast majority of arterial pressure determinations the state of the arterial wall is inconsiderable, whether in healthy or diseased conditions, but that, in some abnormally resistant arteries heightened readings of systolic pressure may obtain.* In these cases the method of preliminary digital compression of the brachial artery to remove resistance, as recommended by the above observers, is, in my experience, of the greatest value in minimising the error of over-estimation

Method of Relaxation of Contracted and Thickened Arteries to secure a Corrected Reading of Systolic Arterial Pressure (*MacWilliam and Melvin* [6])

If resistance is experienced on palpation of the brachial artery, having taken the first reading, the brachial artery is subjected to repeated compression (twenty times), or, preferably, closed by digital pressure with two fingers for three minutes at the middle of the arm. Resistance of the arterial wall, if present, is thus almost entirely overcome, as shown by the second corrected reading, which is now made, being reduced to the level found in a non resistant artery, but not below that level; further compression causes no further reduction

This method does not equalise the arm-leg differences in reading associated with cases of well-marked aortic incompetence,[6] in which there is no correspondence between the degree of pulse pressure and the arm-leg difference.[42]

The Differential Blood Pressure Sign.—This term has been applied by Dr. E. F. Cyriax [43] to differences between the systolic blood pressure readings in the two arms. Such differences may be—

(*a*) *Anatomical*, where the arteries of the two limbs differ in size and distribution.

(*b*) *Physiological*, where the first measurement is heightened by psychic or physical causes, the effects of which have passed away by the time that the other arm is examined. If the patient lies on one side, the maximum pressure in the arm lying beneath is usually slightly higher than that in the arm above.[44]

(*c*) *Pathological*, where the two sides manifest differences as the result of aortic or other aneurysm, intrathoracic new growth, arteriosclerosis involving especially that portion of the aortic arch from which arise the innominate and left subclavian arteries, hemiplegia, or a cervical rib. In unilateral war traumatisms E. F. Cyriax [43] has shown that the systolic pressures in the upper limbs are not always identical, and that the pressure in one arm may be higher than in the other on one day and lower a few days later. In 819 observations on the blood pressure in the two arms

in seventy-three cases of unilateral or bilaterally unequal conditions produced by trauma and operation, together with a few of bilaterally equal operations and operations in the middle line, the differential blood pressure sign is nearly always present. For maximum pressures differences of 10 mm. were found in 83 per cent., and of 20 mm. or over in 12 per cent., of all cases; for minimum pressures similar differences were found in about 80 per cent. and 20 per cent. In a second similar investigation, comprising 1,807 observations on 128 cases, in all the differential blood pressure sign was present.

E. F. Cyriax[45] believes that heightened blood pressure is often due to such irritative states of the spinal extensor muscles as hypertonicity, diffuse fibrosis or fibrositis and venous congestion, which are stated to produce a continuous series of sensory stimuli to the posterior spinal nerves, *i.e.*, a never-ending series of pressor effects which can be successfully treated by appropriate muscle kneading and other movements.

The results of 160 bilateral readings taken by Dr. R. J. Cyriax[46] in twelve patients with pulmonary tuberculosis are comparable, and will be found mentioned on p. 227.

When due to abnormal local conditions of the arterial wall, in a minority of cases the higher readings may be notably reduced by continued local compression, as already described, and in this way divergent readings may be harmonised, the same readings being then obtained from all the limbs. Comparison with a second reading from the other arm, which has not been subjected to repeated or continued compression, is essential, as the pressure may have altered during the period of estimation. No reduction in pressure is found in the absence of abnormality in the arteries.[2]

CHAPTER III

Present-day Instruments Employed in Estimation of Arterial Pressure

BLOOD PRESSURE INSTRUMENTS TO BE RECOMMENDED

The medical man who is about to purchase a sphygmomanometer naturally is inclined to ask what instrument is the most practical and will give him the best return for his outlay.

In Great Britain and America two types of instrument are in common use: A. mercurial, B. dial aneroid. On the Continent oscillometric aneroids C. are largely employed.

A. Mercurial Sphygmomanometers

No matter what claims are put forward by enthusiastic inventors or manufacturers of aneroid apparatus, it is certain that a properly constructed mercurial manometer is capable of registering pressures over long periods of time with greater accuracy and reliability than any instrument that depends upon the elasticity of metal. This does not mean, however, that the *average* mercury manometer used in practice is necessarily more accurate than an aneroid. The majority of mercurial sphygmomanometers in daily use till recent times have a narrow U-tube, along the distal arm of which is fixed a scale bearing equal divisions intended to read in millimetres of mercury. These scales, being stamped by a machine, make no allowance for variations which occur in the calibre of the glass tube at various levels. For the reason that it is a matter of great difficulty to blow a fine glass tube of uniform bore, no two U-tubes are likely to give similar readings on a machine-made scale. This is the first source of inaccuracy. A second

is found in capillary action of the mercury column. When tubes of fine bore are employed, the mercury, especially if at all dirty, tends to adhere to the glass, and gives readings which are inconsistent. A third possible source of error arises from the smallness of the intervals between the divisions on the measuring scale, which thus becomes difficult to read with precision, except in a clear light and by those who possess good eyesight.

The first of the above disadvantages can be overcome by individual calibration of each manometer tube against a standard, or by obtaining after repeated trials a tube of even and uniform bore throughout. The second and third disadvantages are alike obviated by making one arm of the U-tube shorter and of considerably larger diameter than the other, thus forming a reservoir. In this event the ratio between the diameter of the reservoir and that of the small arm of the tube must be accurately determined, and the scale attached to the latter must be compensated for any fall in level of the mercury in the reservoir. Manometers made on this principle are superior to those formerly employed in that glass tubing of three or four times larger diameter can be utilised, with the result that a much more open and clear scale becomes necessary to register the greater amplitude of range of the mercury column through given changes of pressure. The graduations of the measuring scale are thus larger and much easier to read, and because of the wider bore of the tube clean mercury no longer sticks.

The Baumanometer (Figs. 1, 7, 8).—This is the only mercurial sphygmomanometer within the author's experience which comprises all the foregoing mechanical advantages. In simplicity of construction and accuracy of registration it constitutes a distinct advance upon previous types of instrument, from which it differs in the size of the long arm of the U-tube, whose calibre is about four times larger. The shorter arm of the U-tube is expanded into a large standardised steel reservoir, the ratio of its bore to that of the long arm being accurately known. A scale, measuring in calibrated millimetres, each small division of which reads to 2 mm., and the larger divisions in tens, is engraved by hand directly on each accurately interchangeable glass

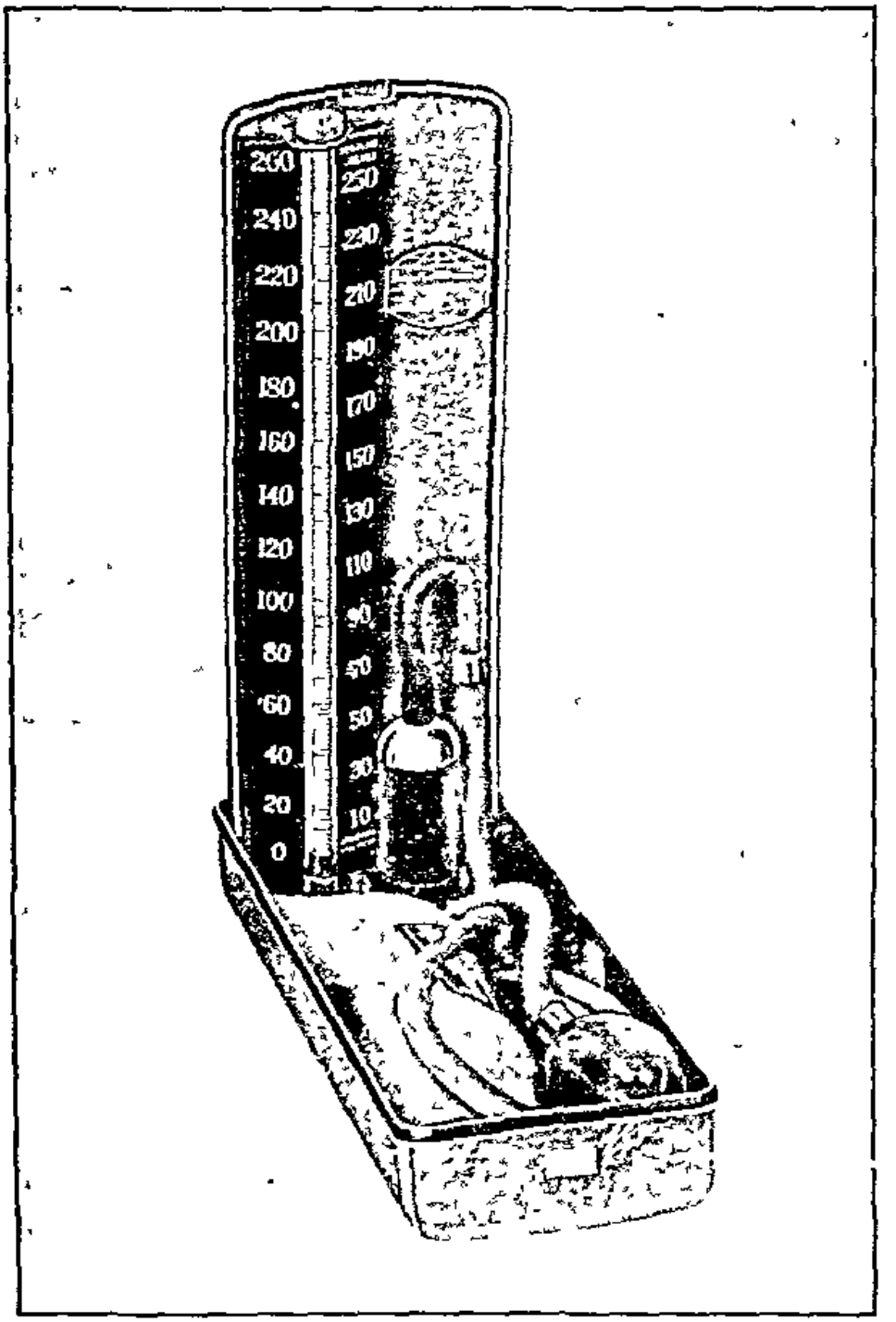


FIG. 7.—The Baumanometer ("Kompak" model) reading from 0 to 260 mm. On the grounds of accuracy, simplicity, permanence, portability and strength of construction this form meets the requirements of most general practitioners The case is of cast duralamin. Size $1\frac{5}{8} \times 3\frac{1}{4} \times 11\frac{1}{8}$ in. Weight 30 oz.

tube, the figures in tens corresponding to the calibrations being placed alternately in white on a black background on each side of the readily cleanable tube. The scale reads from 0 to 300 mm. in the full-sized models, and is adjusted so that the level of the mercury in the reservoir and in the long arm of the U-tube when at rest stands at 0.

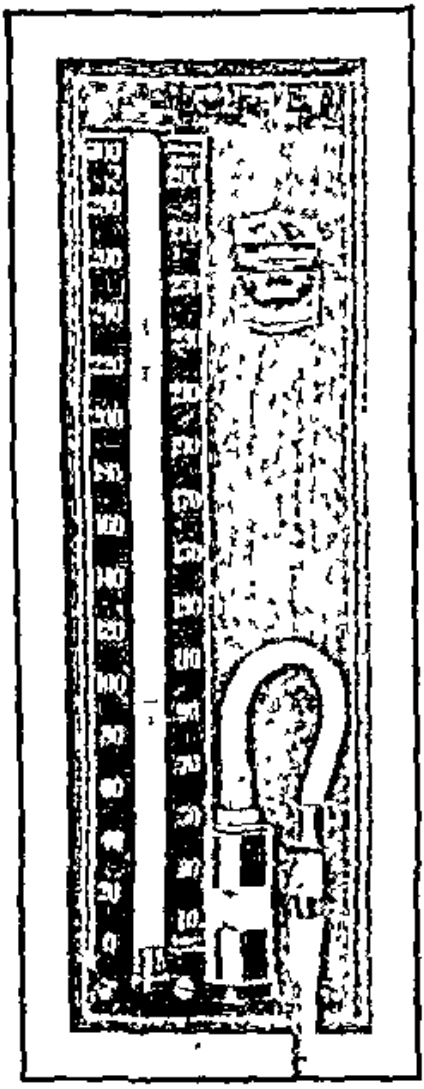


FIG. 8.—The Baumanometer Wall model suitable for use in hospital or consulting room (replacing the former cabinet model) reading from 0 to 300 mm. Size 4½ × 14 in., with 6 ft. of tubing

The makers claim that every instrument is individually calibrated against a standard mercurial manometer, the accuracy of which has, in turn, been checked against the U.S. Bureau of Standards manometer at Washington, *i.e.*, that the scale of each Baumanometer is graduated by hand, so as exactly to compensate for any inaccuracies of the particular glass tube to which it is fitted.

Description of the Instrument (Figs. 7, 8).—To the open end of the long arm of the U-tube is screwed a small metal cap perforated with three small holes and containing a diaphragm. This device, whilst ensuring that the mercury cannot spill, allows air to pass into the tube, so that an even air pressure is maintained; otherwise the instrument would act as a compressed-air manometer and the mercury readings would be too low. To prevent the mercury escaping from the reservoir, a fine glass tube is sealed into its upper end, which is drawn out into a tube for attachment of the heavy walled pure rubber tube leading to the pressure bag. The latter is encased within a satin armlet and has

connected to it a shorter length of similar tubing, which ends in a rubber bulb for purposes of inflation fitted with a sensitive release-valve.

The whole apparatus is attached to the spring-lid of the case, and can be inverted without risk of the mercury being split.

Method of Employment.—1. Turn the milled knob on the outside of the case in a clockwise direction. With the other hand resting against the lid, allow it to swing gently upright. *Do not let the lid fly up suddenly.*

2. Hold the glass tube near the top with one hand, and unscrew the small metal cap with the other, but do not detach the diaphragm from the inside of the cap.

3. Carefully pour the mercury from the bottle sent with the instrument into the U-tube by means of a glass or paper funnel. Should any mercury be lost, place the Baumanometer on a level surface and add enough mercury to bring the rim of the meniscus even with the figure "0" on the scale. To judge this correctly, the eye must be on the same level as the "0."

4. Screw the metal cap firmly in place. The Baumanometer is now ready for use.

5. In order to obtain greatest accuracy, it is important to tap the instrument, so as to ensure that the mercury takes up its proper position, and to stand the instrument on a level base. Errors due to incorrect levelling may be checked by examining the zero reading before putting the sphygmomanometer under pressure.

6. The further procedure is given in detail on pp. 25 to 29 (Fig. 1).

The above description refers to the standard desk-model. Larger and smaller sizes are also made. One defect common to the full-sized instruments as well as to other makes of mercurial sphygmomanometer is that they register only up to 300 mm. of mercury. The "Kompak" and pocket Baumanometers are still more restricted in range, reading as they do only up to 260 and 200 mm. respectively.

The latter instrument, however, is designed for the special purpose of examinations for life assurance, any patient whose arterial pressure reaches the 200 mm. limit being at once rejected.

Whilst it is true that pressures of over 300 mm. are very rare, nevertheless these do occur, and for purposes of general utility one should be able to measure them accurately, which is impossible until manufacturers of arterial pressure instruments realise that a limited scale, while covering ordinary ranges of pressure, is not capable of assessing extremes. At present, utility is subordinated to portability.

B. Dial Sphygmomanometers (Aneroid)

Of portable aneroid instruments suited for clinical work the improved Lauder Brunton apparatus and the Tycos are largely used in this country and in America. Various other forms of dial instruments with single broad inelastic armlet are employed in France, each of which possesses individual features. An early model was the Vaquez-Laubry sphygmotonoscope.[47] In current use are Boulitte's sphygmomanometer, the sphygmophone of Boulitte-Korotkow, and the arteriotensiometer of Donzelot,[48] all designed to register maximal and minimal pressures.

In the use of any of these instruments care should be taken that they are not inclined, since owing to lack of any interior balancing mechanism different readings will be given if the aneroid is sloped

1. The Brunton Sphygmomanometer (Fig. 9).—This apparatus, suggested by the late Sir T. Lauder Brunton, F.R.S., comprises (*a*) a pressure gauge resembling an aneroid barometer, the dial of which (only 2 inches in diameter) is graduated in millimetres of mercury by comparison with a "standard" mercurial manometer, and registers from zero to 300 mm., (*β*) a broad armlet, the rubber bag of which measures 13½ inches by 4½ inches; (*γ*) a metal inflating pump with release screw

In modernised form, the dial gauge made by Jaquet of Basle possesses considerable accuracy. Great sensitivity is imparted to the movement by a double-acting pressure chamber and a specially constructed transmission.

Directions for Use.—Apply the armlet evenly to the arm. See that the screw of the T-valve is close home. Slowly raise the pressure in the armlet by the pump until the radial pulse

can no longer be felt. Then allow the air to escape gradually by reversing the T-valve screw, and observe the pressure

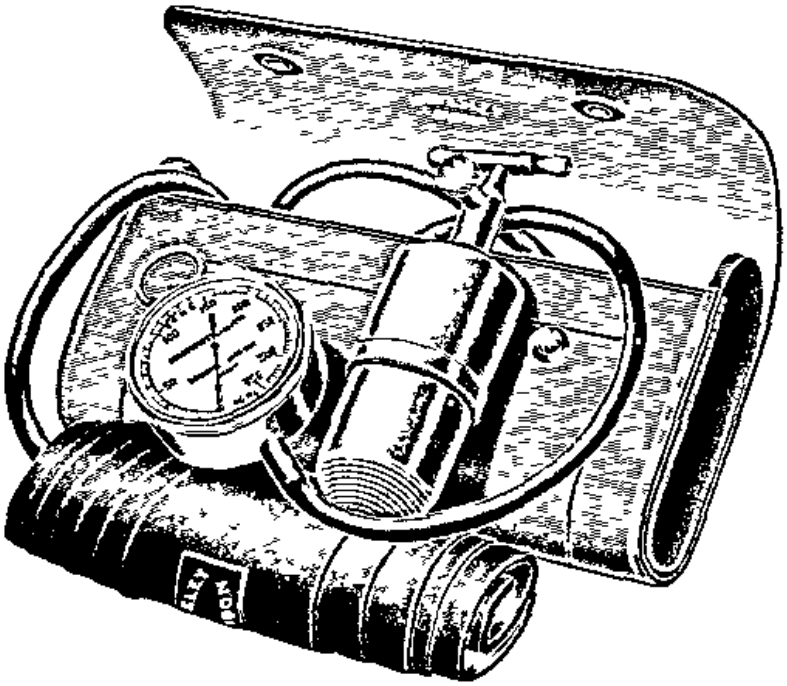


FIG. 9.—The Improved Brunton sphygmomanometer

indicated on the scale at the time the pulse returns. This indicates the systolic pressure. The dial gauge, having a movable scale, can always be brought to the zero point before use.

2. The Boulitte Sphygmomanometer (Fig. 10) is the French equivalent of the American Tycos, over which it possesses

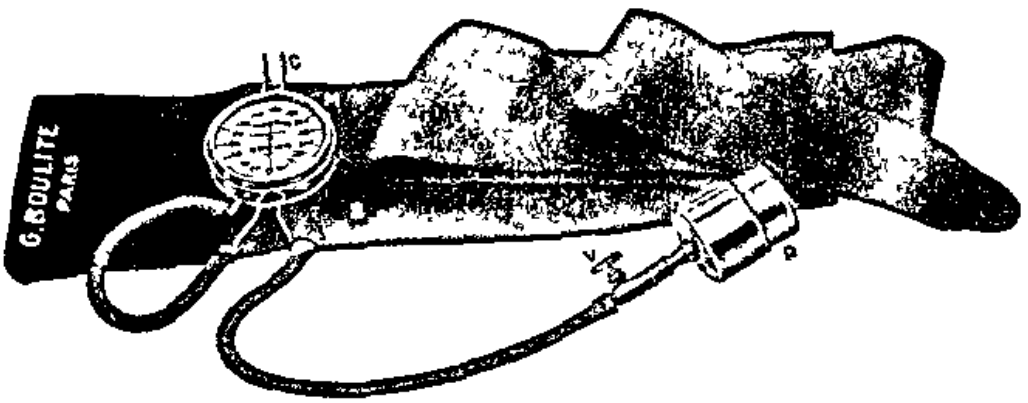


FIG. 10.—The Boulitte sphygmomanometer.

several advantages. These are a metallic pump; an easily adaptable silk sleeve of considerable length, which can be

wrapped several times round the arm; and a dial-needle which, not being checked, permits independent control. The instrument is very light and handy. It is provided with a leather case which goes easily into the pocket.

3. The Tycos Sphygmomanometer.—This instrument,

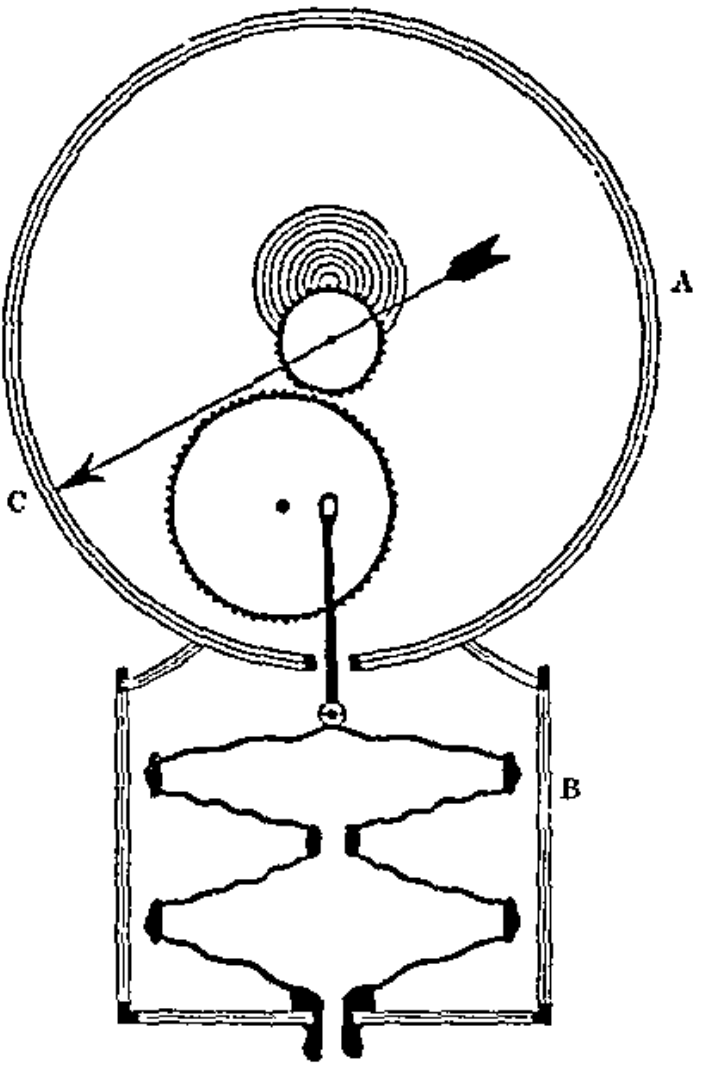


FIG. 11.—Diagram showing the internal mechanism of the Tycos aneroid sphygmomanometer (Norris) A Dial case B Metal case beneath dial containing aneroid chamber composed of two metal discs connected by a lever affixed to a cog wheel, which engages with a smaller cog wheel and so causes movement of the dial needle, C.

originally devised by Dr. Oscar H Rogers, is popular with the general practitioner chiefly because of its compactness and portability (Figs. 11, 12, 13). Two accurately ground metal discs are attached at their margins to the metal case beneath the dial. These discs constitute an aneroid

chamber, into which air can be pumped by the bulb, thus slightly separating them; with the highest pressures only 2 to 3 mm. of expansion occurs, the measure of resistance of the chambers being accurately determined. A lever affixed to a cog, in turn connected with the dial needle, greatly magnifies the slightest expansion of the discs (Fig. 11). Each dial is made by hand, and the scale divisions on it are graduated against a standard mercury manometer, so that no two dials are exactly alike as regards spacing of the

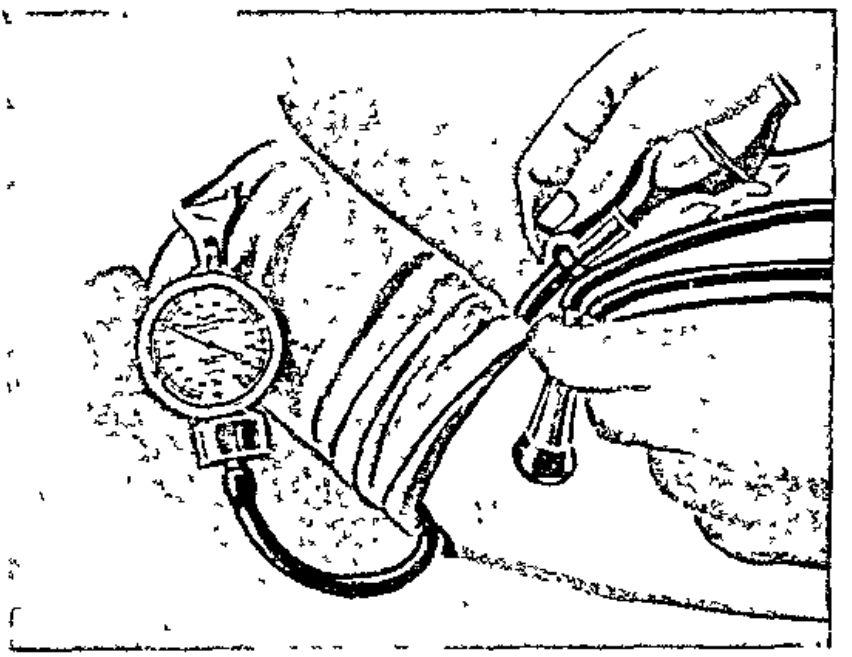


FIG. 12.—The Tycos aneroid sphygmomanometer employed in combination with the auditory method.

divisions. The relation of the needle to a zero point in the middle of an oval on the dial furnishes a check on the accuracy of the instrument. The dial is graduated up to 300 mm. Hg, and the 20-mm. spaces on it are accurately subdivided into ten equal scale divisions, each representing 2 mm. of pressure. The needle travels round the dial, so that its movements can readily be followed. In releasing the pressure these movements are visible before the sound can be heard, and are to be disregarded. They are simply due to beating of the blood against the upper edge of the armlet.

The instrument is unaffected by changes in temperature and barometric pressure, and, with reasonable care and occasional cleaning, should last for several years. At the end of every two years it should always be tested against a standard mercury manometer, since by this time it often

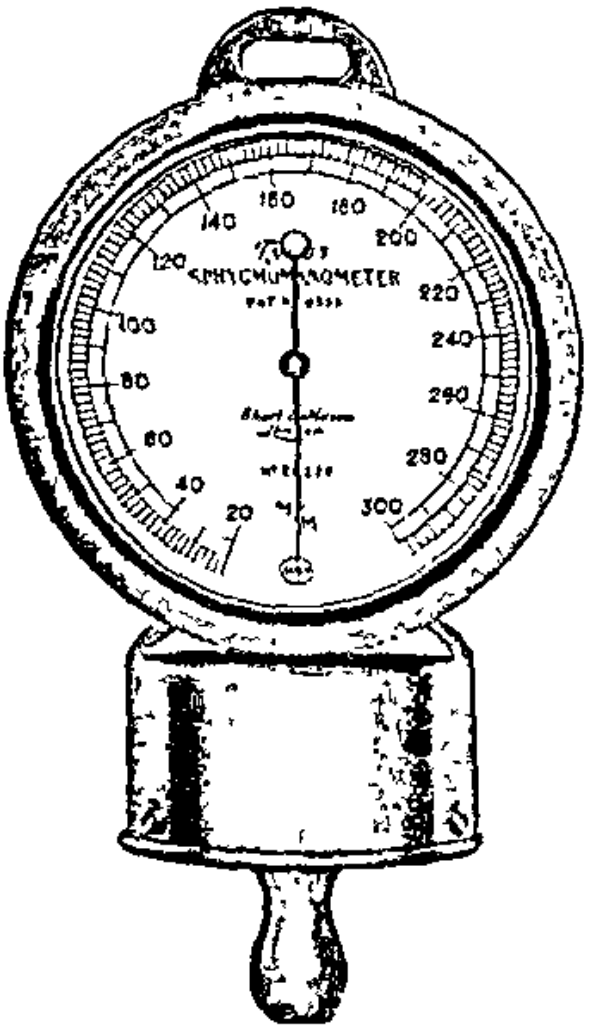


FIG 13.—The Tycos sphygmomanometer, exact size

needs readjustment, and is then liable to give readings which are too low in the case of high arterial pressures.

4. The Arteriotensiometer of Donzelot[48] (Fig. 14), designed for rapid, easy and precise measurement of arterial pressure, is well adapted for modern work. The cuff is inextensible, and is readily adjusted by means of two bands of webbing, each of which passes through a metal fastener, which clips and secures them tightly. The rubber air-bag

is covered with silk, which produces no sensations of cold to the arm of the subject. The manometer is strongly made, and practically indestructible. It is graduated in millimetres of mercury, and allows estimation of maximal and minimal pressures to within 2 mm. No check is placed on the needle, which, at rest, freely returns to the centre of the oval, denoting zero, so that precision can thus be assured. By reason of this, the manometer is self-controlled, whereas, in ordinary manometers, a check being applied to the needle before its

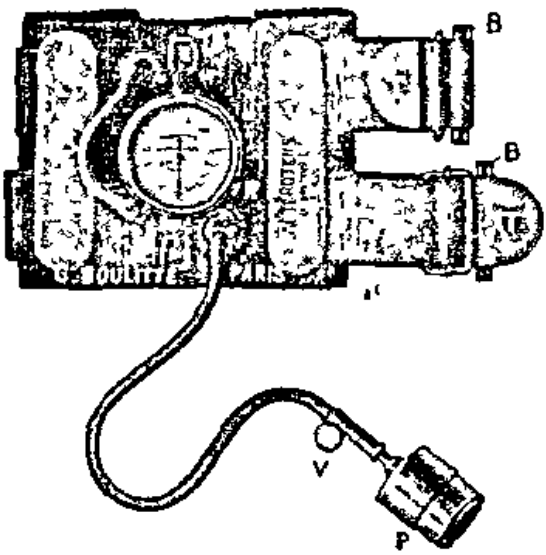


FIG. 14.—The arteriotensiometer of Donzelot.

position of rest may mask an error in the apparatus. A regulating button is added, by which any faulty position of the needle can be regulated as easily as the hands of a watch. The pump is of metal, and, because of its durability, superior to indiarubber bulbs. The escape valve is wedge-shaped, without intervention of leather or rubber, which ensures accuracy and sensitiveness, while a safety-catch prevents the screw from being opened too wide and thus lost. The stethoscope supplied with the instrument is of modern construction.

C. Oscillometric Sphygmomanometers (Aneroid)

1. The Sphygmo-oscillometer of Pachon.[49, 50]—The sphygmo-oscillometer (Fig. 15) consists of a cylindrical air-

tight metallic box which contains an aneroid capsule of aluminium. The rigid box, the contained aneroid chamber and the armlet communicate directly with one another by means of a three-way channel. The pressure within this system at any given moment is indicated on the dial of a small manometer graduated from 0 to 35 cm. of mercury. A separator key, on being depressed, closes the transverse limb of the three-way channel, and so allows pulsations set

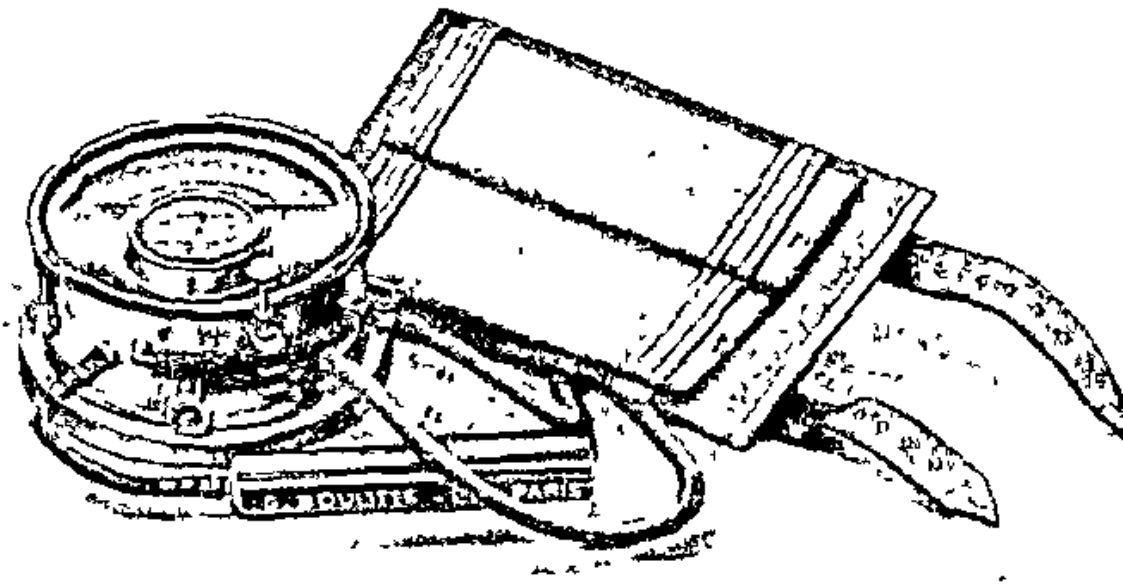


FIG 15.—The Pachon sphygmometric oscillometer (new model) showing Gallavardin's armlet with two independent pressure bags p^1 and p^2, one of which overlaps the other by about one third of its diameter; t^1 and t^2 = pressure tubing connecting the oscillometer box with the pressure bags p^1 and p^2 respectively, r = release tap to p^1, s = separator key, v = screw valve for release of external pressure

up in the armlet to be transmitted direct to the interior of the aneroid chamber, and from this to the delicate registering needle. In use the instrument is connected, by means of a single narrow armlet with the *wrist* (Pachon), or by means of a broad double brachial armlet with the arm (Gallavardin). The pressure throughout the instrument is raised by means of the pump to a point above the maximal pressure of the subject. The separator key is then depressed and the registering needle watched. If no movement of

the needle occurs, the pressure should then be gradually lowered by opening the screw valve.

Note—The separator key must never be touched while the screw valve is open, lest sudden decompression should injure the aluminium capsule, so that, in order to avoid the possibility of accident, it is advisable to use the right hand only to manipulate escape valve and separator key alternately.

The first of a series of **increasing** *oscillations of the registering needle indicates the maximal pressure,* S (Fig. 5). Before this point is reached small deflections of the needle, differing

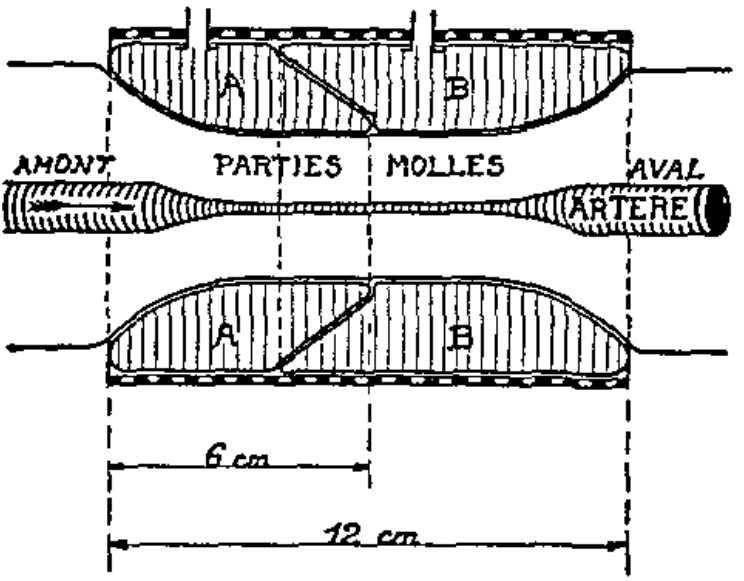


FIG. 16.—Represents diagrammatically in section Gallavardin's armlet inflated upon a limb. The artery is compressed uniformly as a result of the overlapping of the two bags.

little from each other, not infrequently occur (supramaximal oscillations, Fig. 5), especially in high pressure cases, but these are to be disregarded, and only the larger one, which constitutes the beginning of the zone of increasing oscillations, is to be noted. The pulsation which precedes this point and the one which follows it serve as controls.

As the pressure is further lowered the oscillations gradually increase in size, thus constituting a second zone distinct from the first. These large oscillations pass progressively to a point at which they attain their greatest amplitude,[25, 26, 27] the index of the "efficacious" or mean pressure,[56] afterwards decreasing in regular or irregular fashion to terminate

in a third zone of oscillations, only slightly differentiated from each other, the onset of which zone corresponds with the minimal pressure D (Fig. 5).

During the phase of gradually increasing oscillations, by watching the excursions of the needle, one can study the rate, rhythm, form and amplitude of the pulse. This in itself constitutes one of the distinct advantages of the oscillometer, since by its use arrhythmias of respiratory or juvenile type, premature contractions, paroxysmal tachycardia, and auricular fibrillation, etc., can readily be observed. The number of divisions on the oscillometer scale traversed by

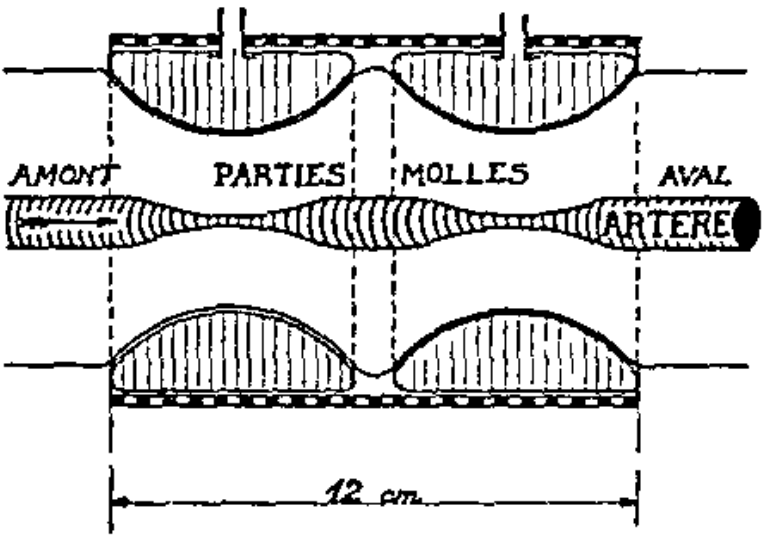


FIG. 17.—Represents diagrammatically in section how the artery is subjected to discontinuous double compression by two inflated bags placed side by side in the same armlet, but without overlapping. (Gallavardin.)

the needle in its widest excursion gives the pulse wave; *e.g.*, if the needle makes its greatest swing between 4 and 10, this represents six divisions on the scale.

Pachon's model has a pressure bag of 7·8 cm. in width, which is applied just above the styloid process of the radius. In consequence of this narrow armlet the readings are always higher than those of the standard mercurial manometer, the auscultatory readings of maximal pressure being from 10 to 20 mm. higher, but the minimal much the same.

Following Gallavardin's [51] suggestions, these higher readings, which, however, Pachon still holds to represent correct pressure values, have been brought into line with other instrumental records in later forms of oscillometer by

the use of two separate pressure bags in the same cuff, which overlap by about one-third of their width, and have a combined width of 12 cm. (Figs. 16, 17, 18). It can easily be demonstrated that these two bags actually do exercise uniform compression upon an artery by inflating the armlet over an empty bottle and seeing how the whole inner surface of the two bags constitutes a perfect cylinder with a very narrow circumferential line of demarcation between them. Each is connected with the oscillometer by pressure tubing, an air release being inserted in the circuit of the upper pressure bag. This arrangement allows

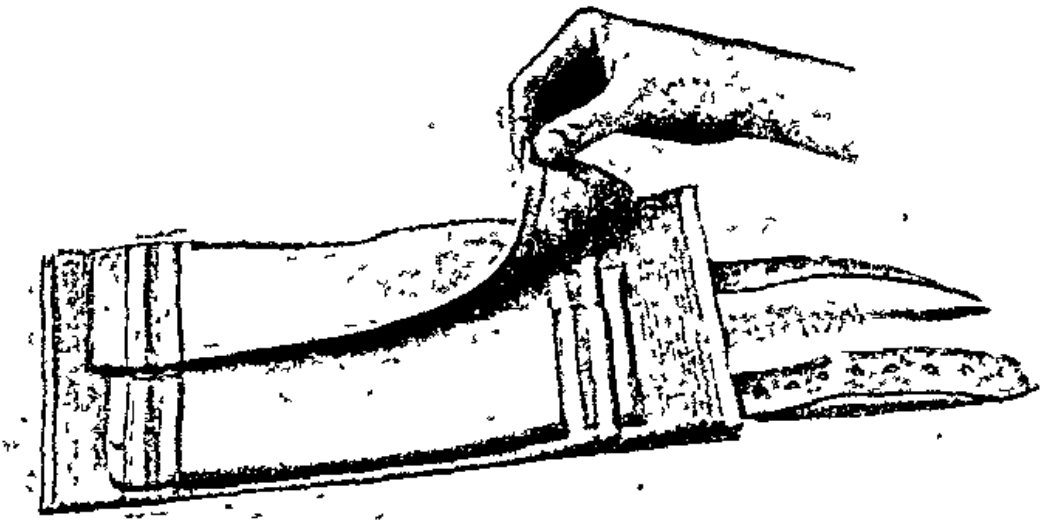


FIG. 18.—Gallavardin's armlet with two independent pressure bags, which overlap by about one-third of their width and have a combined width of 12 cm.

the observer to record separately the oscillations which are produced under the central portion and under the peripheral portion of the same armlet. The blood pressure is estimated in the brachial artery, instead of at the wrist with Pachon's former model of instrument, and gives readings comparable with those of the standard mercurial manometer.

2. Boulitte's Universal Oscillometer (Fig. 19).—This apparatus, an improved form of the older Pachon, is highly to be recommended both for its mechanical and physical qualities as well as for its clinical utility. It fulfils all requirements for precise estimation of minimal, maximal and mean pressures, is of much reduced size and weight, strongly made,

easy to manipulate, and difficult to derange. The needles of the manometer and of the oscillometer are each provided with a regulating screw, so that in the event of either needle assuming a faulty position, as the result of a fall, for example, it is not necessary to send the apparatus back to the makers. Friction is reduced to a minimum, and lag is absent. Hence the needle traces with fidelity the pulsations communicated to it, the amplitude of these under different conditions being exactly proportional to the strength of beat. Each instrument being of equivalent sensitiveness, accurate determina-

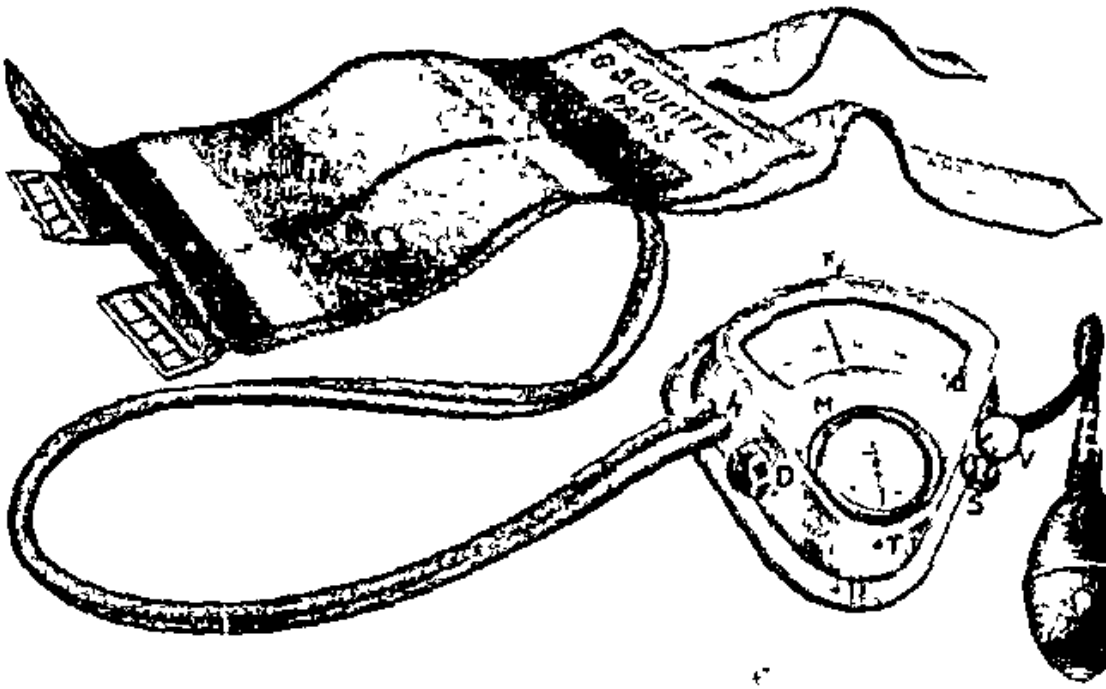


FIG. 19.—Boulitte's universal oscillometer with Gallavardin's armlet.

tions and true comparisons can be made of arterial pressures in the investigation of arterial permeability and elasticity. The apparatus is universal in that it can be applied to measurement of arterial pressure by any of the ordinary methods—oscillatory, auditory, vibratory or tactile. It is carried in small compass within a neat oblong leather case, without detaching the rubber tubes, so that it is always ready for service. In short, it is a good sphygmomanometer with many supplementary advantages.

3. The Kymometer of Vaquez, Gley and Gomez (Fig. 20) — These authors attach considerable importance to determination of the mean dynamic pressure in clinical medicine.[52]

They have, therefore, devised this special oscillometer furnished with a new contrivance by which the oscillometric needle always starts from the same fixed point. Thus in reading amplitudes the point marking the *extent* of the excursion is all that matters.

With this apparatus the reading of the greatest oscillation (oscillometric index) characterising the level of the mean pressure is rendered very simple.

Internal Mechanism.—The kymometer, in common with all the other oscillometers (universal, portable and arterial) made by Boulitte, consists of an aneroid capsule C enclosed

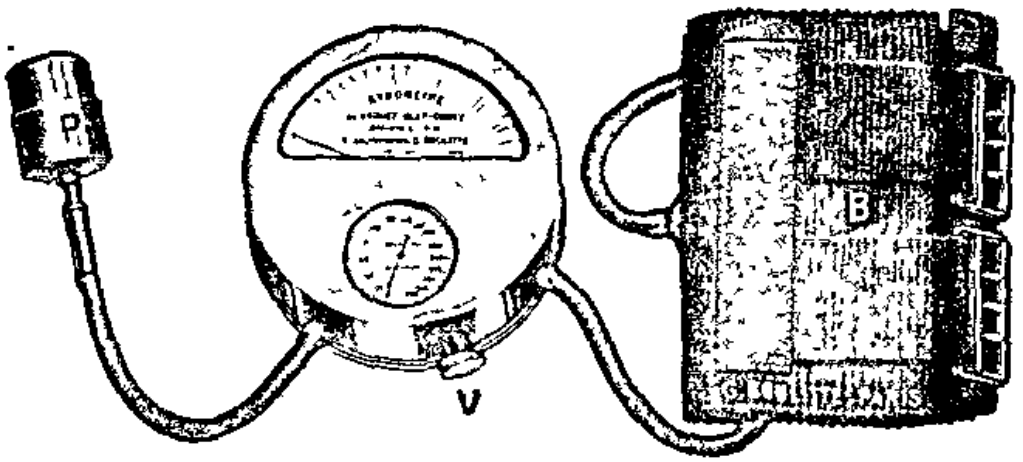


FIG. 20.—The Kymometer of Vaquez, Gley and Gomez.

within a rigid and airtight metallic box. The capsule is put into communication with the box by means of a valve S, so arranged that air can pass from box to capsule, but cannot pass from capsule to box (Fig. 21). Further, the capsule is connected with the cuff B by a two-way tube, one limb of which carries the escape screw V. A metallic pump P allows air to be driven into the box, the pressure within which, as well as that within the capsule, is indicated by the manometer M. This equality of pressure constitutes the chief feature of the apparatus.

When air is pumped into the box, this air pressure opens the valve S and gains the capsule. If, on the other hand, air is allowed to escape from the capsule by the escape screw V, the valve again opens and the equilibrium of pressure between the box and capsule is maintained. When the

escape screw is shut, and arterial pulsations distend the capsule, the valve then remains shut and the pulsations are transmitted to the delicate oscillometric needle. The valve, being extremely light but very solid, acts as an automatic separator (replacing the somewhat troublesome separator key in the older Pachon). At whatever moment V is shut

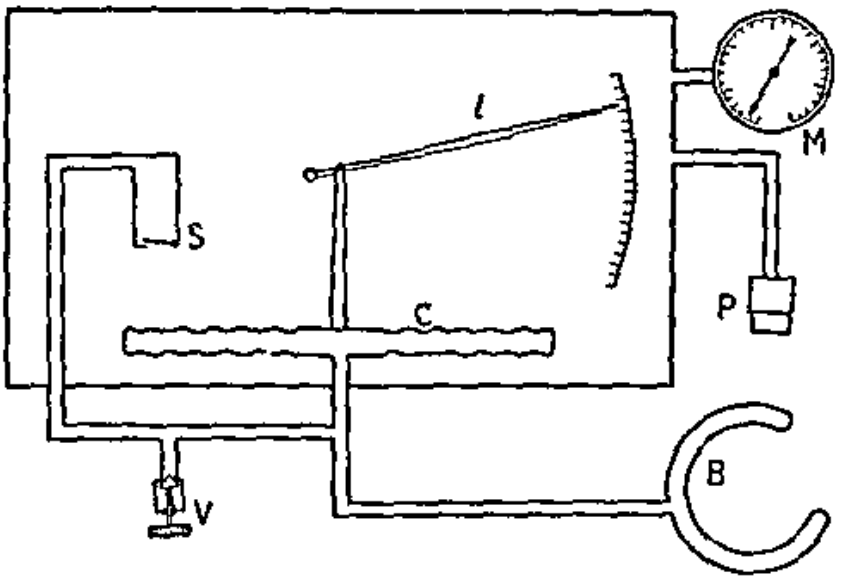


FIG. 21.—Diagram illustrating the internal mechanism of the universal and other sphygmo oscillometers. (By courtesy of M. G. Boulitte.)

The rigid and airtight metal box contains an aneroid capsule, C. The cylindrical metal box, the aneroid chamber C, and the armlet B communicate directly with one another by the three way channel.

Any desired pressure can be obtained through the combined system by means of the pump P, the pressure reading at any given moment being indicated on the manometer dial M, which is graduated in centimetres of mercury. An escape valve, V, allows the pressure to be released at will. The one-way valve, S, closes when the pressure becomes equal inside and outside the aneroid chamber, C thus allowing pulsations set up in the armlet to be transmitted direct to the interior of the aneroid chamber and thus to the delicate registering needle *l*, the free end of which swings over a divided scale and so gives the amplitude of the pulse wave.

in order to read the amplitude, the box is in equilibrium with the lowest pressure existing in the capsule, *i.e.*, corresponding to the diastole. the capsule is then at rest, the needle at zero and oscillations begin always from this point.

After the requisite pressure has been introduced, air is allowed to escape centimetre by centimetre while successively opening and closing the escape screw.

Records can be made simply and quickly of (*a*) maximal

blood pressure ; (*b*) minimal pressure ; (*c*) amplitude of pulse wave ; (*d*) pulse rate and characteristics, expressed in four sets of figures, or by means of a chart which furnishes a record of the amplitude of oscillation at different pressure levels.

The Oscillometric Curve

By an oscillometric curve is meant a diagram of pulsations of a limb or arterial segment subjected to a variable range of progressively increasing or decreasing external counter pressures. The construction of such a curve is simple. Upon squared paper two straight lines at right angles to each other are drawn, of which one (abscissa) corresponds with the figures for the counter pressure, and the other (ordinate) to the values of amplitude of oscillation read on the scale of the apparatus. In order to allow greater amplitude to the curve, the oscillations are noted on a scale double to that of the pressures. For each degree of counter pressure, upon the squared paper is marked a point situated at the intersection of the vertical line passing through the value of the counter pressure at the moment, and of the horizontal line passing through the figures of the ordinates equal to the size of the oscillations. All the points are then joined by a broken line, which forms the oscillometric curve.*

D. Self-Recording Sphygmomanometers

I. Indirect Clinical.

1. The Tonoscillograph of Plesch.—(*a*) *The Apparatus.* For many years it has been possible to obtain tracings of blood pressure by means of a kymograph. In clinical medicine, however, through lack of any satisfactory objective mode of registration, a numerical formula has had to represent "the complete arterial pressure picture" (p. 27). A distinct advance has now been made by Professor J.

* For further information on this and allied subjects the reader is referred to Pachon and Fabre, "Clinical Investigation of Cardiovascular Function." English translation by J. F. Halls Dally ; Kegan Paul, London, 1934.

Plesch,[53] of Berlin, who, after several years' experiment, has succeeded in perfecting the tonoscillograph (Fig. 22), which, in estimating arterial pressure,[54, 55] affords many substantial advantages as compared with the ordinary types of apparatus. I have done much work with this instrument since 1929, and prefer it to any other recording machine, since it is of such delicacy and mechanical perfection that the tracings obtained suffer no deformation.

Tonoscillograms produced by this apparatus enable

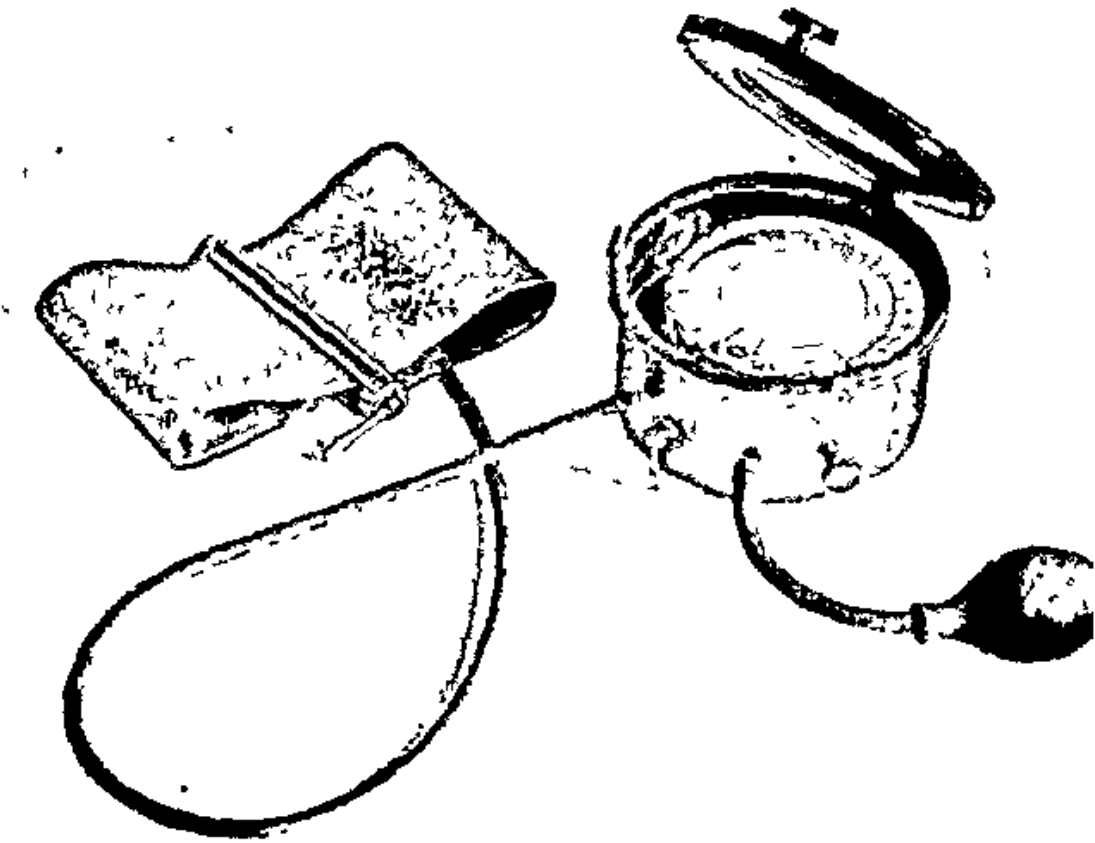


Fig. 22.—The Tonoscillograph of Plesch.

variations in the arterial pressure of any individual to be accurately traced under different conditions. The curves not only yield graphic and permanent records of arterial pressures and of individual pulse characters, but also enable the state of the vascular system to be diagnosed and the oscillations to be measured with a higher degree of accuracy than is possible by any other indirect method.

The taking of an objective tonoscillogram does not occupy more time than is required in the ordinary subjective method

of determining the blood pressure, while the record obtained forms a valuable addition to the clinical chart.

The apparatus is constructed of metal throughout and, being operated automatically by increase or decrease in pressure without any special driving mechanism, is reliable in working and practically free from error.

Like most instruments of great precision, however, the tonoscillograph is delicate in adjustment and requires aptitude in manipulation. Hence, although small and light enough to be portable, it is more applicable for static use in

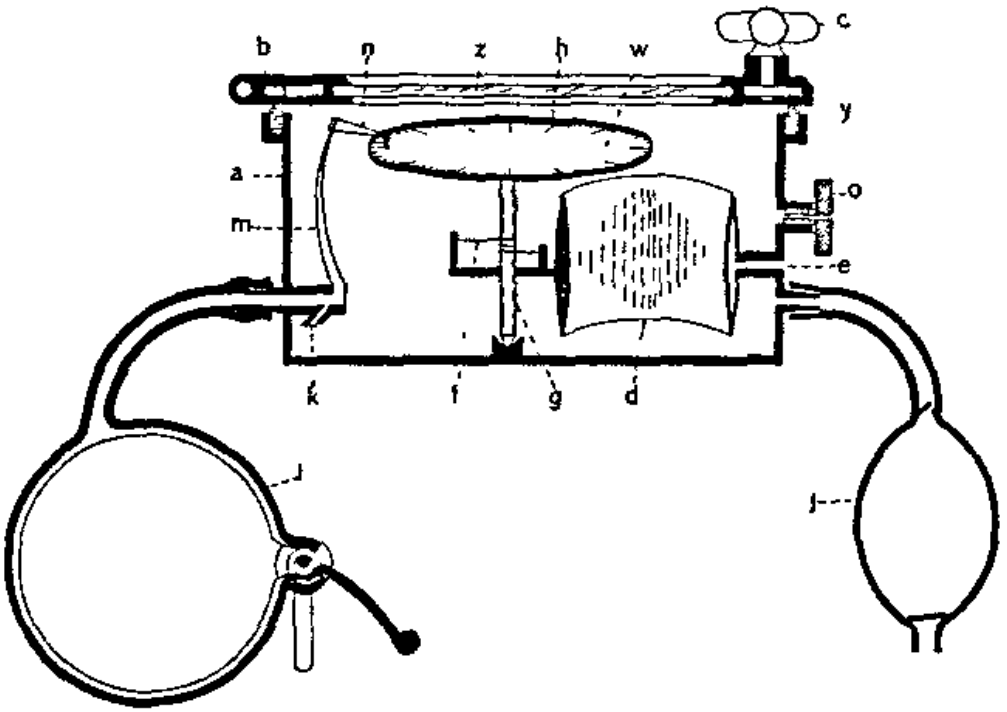


FIG. 23.—The tonoscillograph in vertical section (diagrammatic).

the consulting rooms of those who make a special study of circulatory phenomena than for daily transport on a round of visits to patients.

Fig. 23 is a diagram of the apparatus in vertical section. The outer metal case (*a*) is made airtight by the hinged lid (*b*), in which is set a glass window, and is fastened by means of the device (*c*). Inside the case (*a*) are two manometer systems. One of the manometers (*d*) communicates through the aperture (*e*) with the outer air, so that the interior of this manometer remains constantly under atmospheric pressure. When the pressure within (*a*) is increased by the

rubber inflating bulb (*y*) the manometer (*d*) becomes bent, thus causing the thread (*f*) to be unwound, thereby rotating the table (*h*) fixed to the spindle (*g*). The other manometer (*m*) has a capillary opening (*k*) into the case (*a*), and is in direct communication with the armlet (*l*). By pumping air into the case, the armlet simultaneously becomes inflated through the capillary opening (*k*). In this way the thin-walled and highly sensitive manometer tube (*m*) is protected from over-distension. Nevertheless, by means of the capillary opening, equalisation of pressure is secured, so that the external and internal pressures remain the same.

Attached to the manometer tube (*m*) is the writing pen (*n*), which records any movement directly on a graduated paper disc set on the table (*h*). Any increase in pressure caused by the pulse will, therefore, be recorded by this pen before the difference can be equalised through the capillary opening (*k*). The size of this aperture can be regulated by a lever; it will readily be seen that the stronger the pulse and the smaller the capillary opening the larger will be the oscillations recorded by the pen. Should these oscillations become unduly large in the case of powerfully beating pulses, they can be damped by increasing the size of the capillary aperture. The valve (*o*) facilitates even reduction of pressure in order that the disc may return to the zero position. During the time that the pressure is falling the pen (*n*) automatically, and in true accordance with the pulse volume, traces impulses, communicated by means of the armlet, on the disc in the form of a curve, the tonoscillogram.

Fig. 24 shows the apparatus viewed from above At the top is the hinge upon which the circular metal lid surrounding the glass window turns in opening and closing Opposite to this at the foot is the capillary lever mentioned above; to the left of this is a nozzle for the rubber tube connecting with the armlet, to the right an attachment for the inflating bulb: still further to the right a screw-valve for securing even release of internal pressure. The paper disc (25), graduated from 0 to 300 mm. Hg, rests upon a rotating aluminium table (23), the edges of this being represented in the diagram by broken lines Tiny projections around its circumference hold the paper disc flat. Opposite (0) is the

writing pen with angled supports by which it can be raised or lowered on a transverse bridge (31) actuated by the capillary lever. To the left of the bridge is a spring (30) for arresting movements of the table.

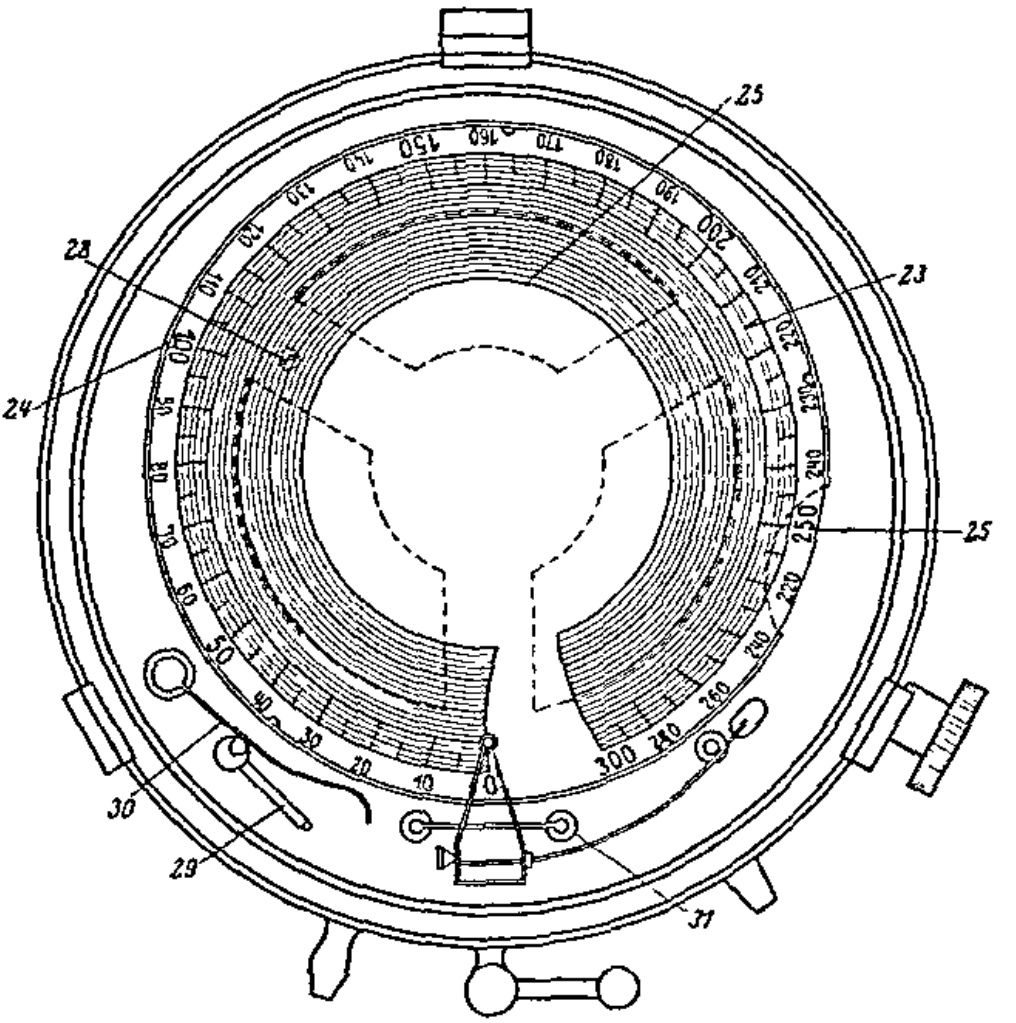


FIG. 24.—The tonoscillograph viewed from above.

Fig. 25 is an analysis of the curve of arterial pressure and volume obtainable with the instrument; the general form of the curve closely resembles those illustrating a paper of mine in 1911 on the sphygmo-oscillometric method of Pachon.[50] As the external pressure is allowed gradually to fall, an approximately even series of small undulations (A) appears as each successive pulse wave impinges against the upper point of arterial constriction. The point for the systolic pressure (S) is marked by the first of a series of slowly increasing waves which, after a varying stage (B) at

the point (P) suddenly become almost double the size of the preceding waves. From this point up to the largest oscillation (C) the increase is much more gradual. Thence onwards the curve is flatter in type, the oscillations slowly diminishing to the point of diastolic pressure (D), at which they undergo

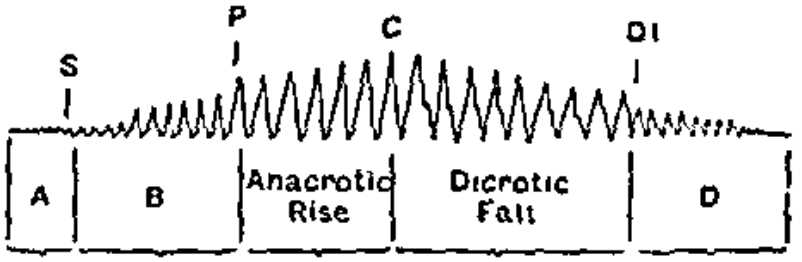


FIG. 25.—The Arterial Pressure Curve, showing successive stages of approach (A), development (B), oscillations (P to D_1), and pulsations (D). S = systolic pressure, D_1 = diastolic pressure. P = point at which pulse waves increase to nearly double the height of the preceding pulse waves, C = mean pressure, *i.e.*, the greatest amplitude of oscillation (Oscillometric Index of Pachon).

a sudden reduction by half. Throughout the whole of the ascending portion of the curve each oscillation is seen to be separated by a flat interval, which becomes less as the highest oscillation is approached. Thereafter the oscillations assume a peak-like ascent. During the ascending portion of the

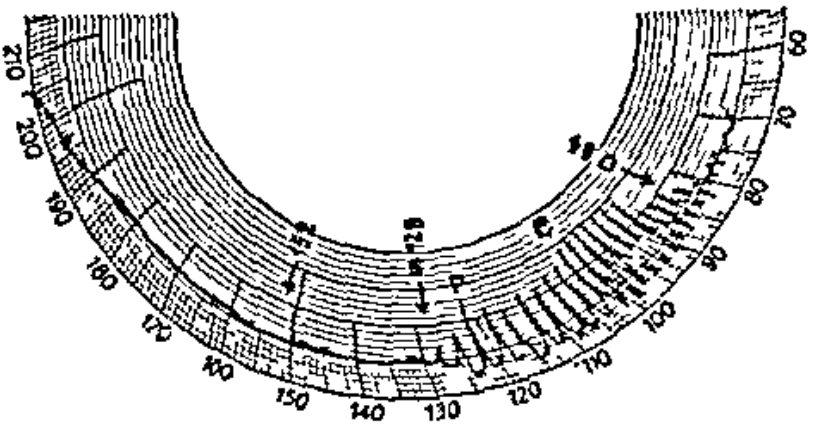


FIG. 26.—Tonooscillogram of healthy man aged 47.

curve the oscillations are anacrotic in form. During the descending portion they are dicrotic, but only as far as the point of minimal pressure. Beyond this point, the external and internal pressures being equalised, the pulse waves are represented by the stage of pulsations.

The foregoing stages are exemplified in the accompanying curves of arterial pressure and pulse volume taken from cases under my care (Frontispiece and Figs. 26, 27, 28, 29).

In the case of a healthy man, aged 47 years (Fig. 26), the stage of approach extends from 152 mm. Hg. to the point of systolic pressure (S) at 128 mm. Next comes a short developmental stage, denoting healthy tone of arterial walls, which ends at 124 mm. (P), from which point the stage of full pulse oscillations with flat anacrotic waves is continued, the intervals becoming less spaced and flattened at the base as they approach C. Following the ascending portion of

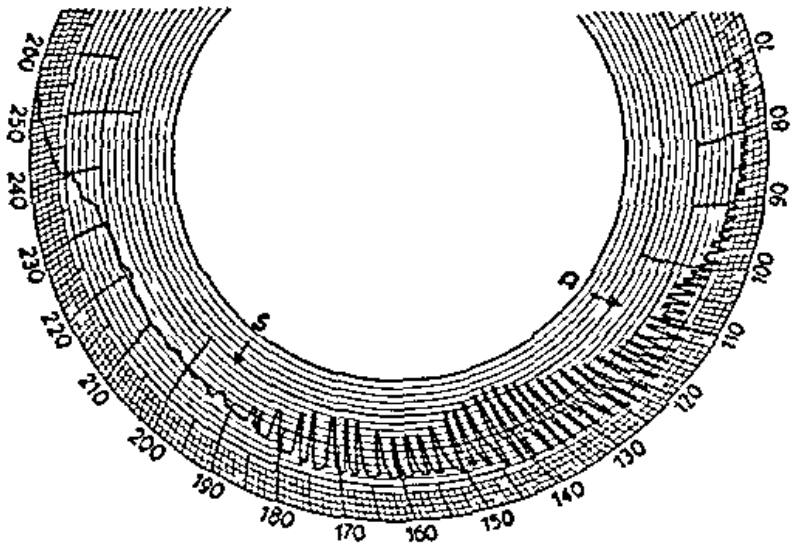


FIG. 27.—Hyperthyroidism.

the curve ending at C, the waves become more uniform in amplitude (descending portion) with evident dicrotism, this ceasing abruptly at 84 mm., the point of diastolic pressure (D). The stage of pulsations completes the curve-analysis.

Fig. 26 is a case of early but typical hyperthyroidism in a lady aged 36, seen in consultation with Dr. R. Tudor Edwards, of Wembley. Herein the various stages of the arterial curve are clearly recognisable.

Fig. 28 is the tracing of a man aged 60 years, 6 feet in height and weighing 15 stone, a teetotaller and non-smoker, athletic in his earlier years and fond of mountain climbing. The chief complaints were of "thumping in the head while in bed," and of pressure feelings referred to the vertex.

The heart showed considerable left ventricular preponderance and a systolic murmur was audible over the whole præcordium with ringing aortic second sound—a case of arteriosclerosis. S is at 194 mm. Anacrotic ascent ends at 150 mm. Descent is gradual to D at 104, where dicrotism suddenly ends. Well-marked pulsations follow this point.

The Frontispiece clearly illustrates high arterial pressure in combination with *Pulsus alternans*. The tracing is of a man aged 47, whose systolic pressure was 269 millimetres of

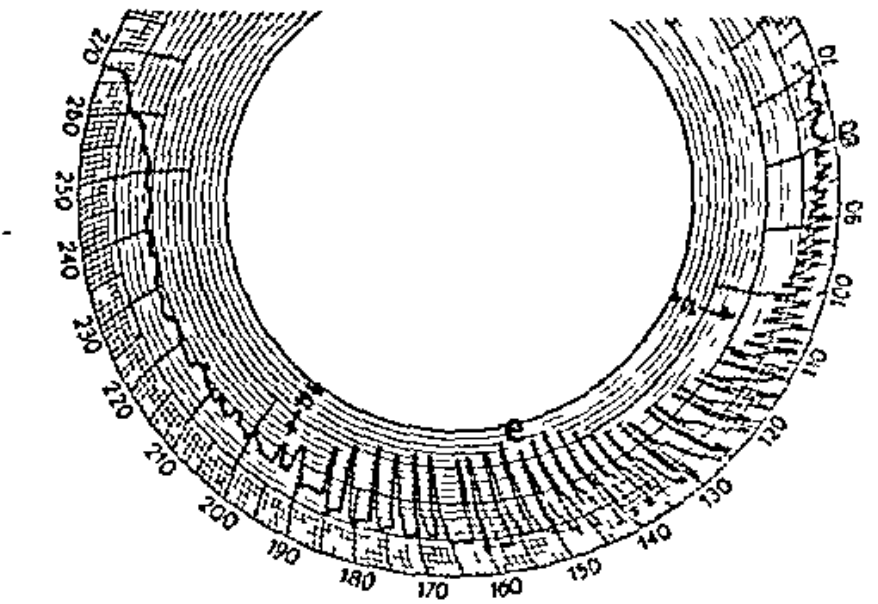


Fig. 28.—Arteriosclerosis.

mercury, diastolic pressure was 151 and mean pressure 204.

In striking contrast to all other curves that I have yet had the opportunity of taking comes Fig. 29, that of a hospital patient, aged 67, housewife, whom I showed in 1925 at the Royal Society of Medicine [56] as a case of hyperpiesia, since established to be of gouty origin.

The later clinical picture was one of cardiac dilatation consequent upon hypertrophy, and changes in the vessels arising independently of renal changes such as occur in the usual forms of Bright's disease. In February, 1930, hæmorrhage occurred in the left retina with impairment of vision two days later. One month after this blurring of speech was noticed, which persisted. The arterial curve is of

unusually bold character, despite the fact that the capillary lever was turned to the full extent so as to damp the amplitude of oscillations. Each pulse beat is seen to be enormous, corresponding clinically with a pulse of full volume and high tension. At every third beat is a regular sequence of premature contractions. The systolic pressure is at approximately 306 mm., but cannot be precisely fixed because this

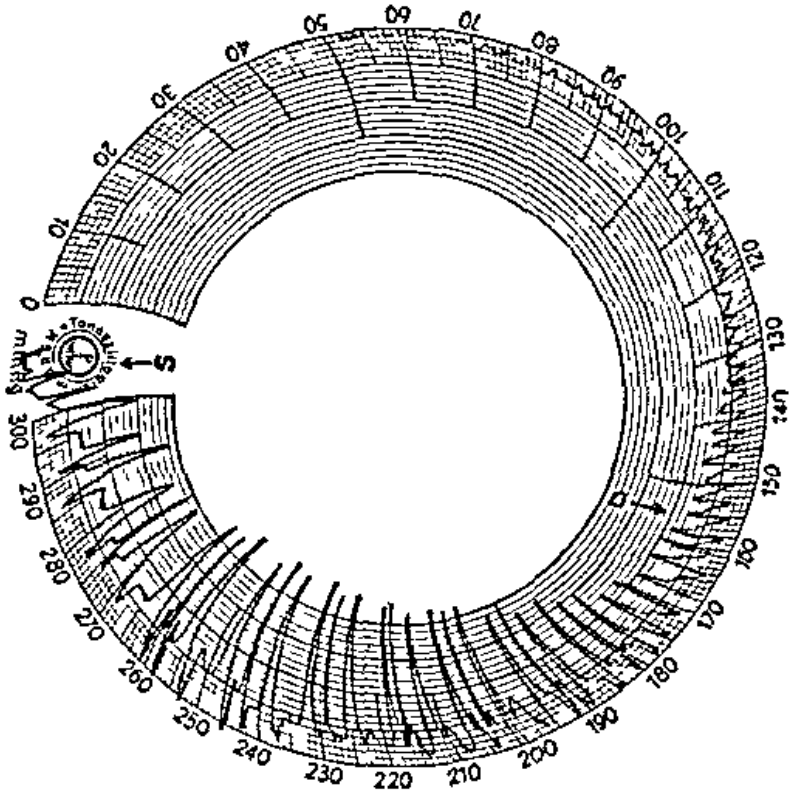


FIG. 29.—Hyperpiesia with secondary changes in heart and vessels.

sphygmomanometer, like the majority of others, has been constructed on the assumption that pressures above 300 mm. Hg cannot occur. This patient's pressure, however, when first seen was 320 mm. systolic, and I have had other cases in my own practice with maximal pressures well above 300 mm. The diastolic pressure is reached at 155 mm., giving a differential pressure of about 150 mm.

2. Boulitte's Portable Recording Oscillometer (Fig. 30).—To Boulitte's Universal Oscillometer (K) is added an oscillographic capsule (C) provided with a pen (J), writing in ink and sensitive enough to record even the weakest pulse. A

clockwork movement (N), regulated by a button (B), propels in a straight line horizontally at requisite speed a roll of paper (E), squared in millimetres.

The record is made in two portions: the first by setting the distributor key at Mn, thus placing in communication the two pockets of the Gallavardin double cuff. This permits estimation of the mean and minimal pressures (Fig 31); the second, by cutting out the upper pocket of the cuff,

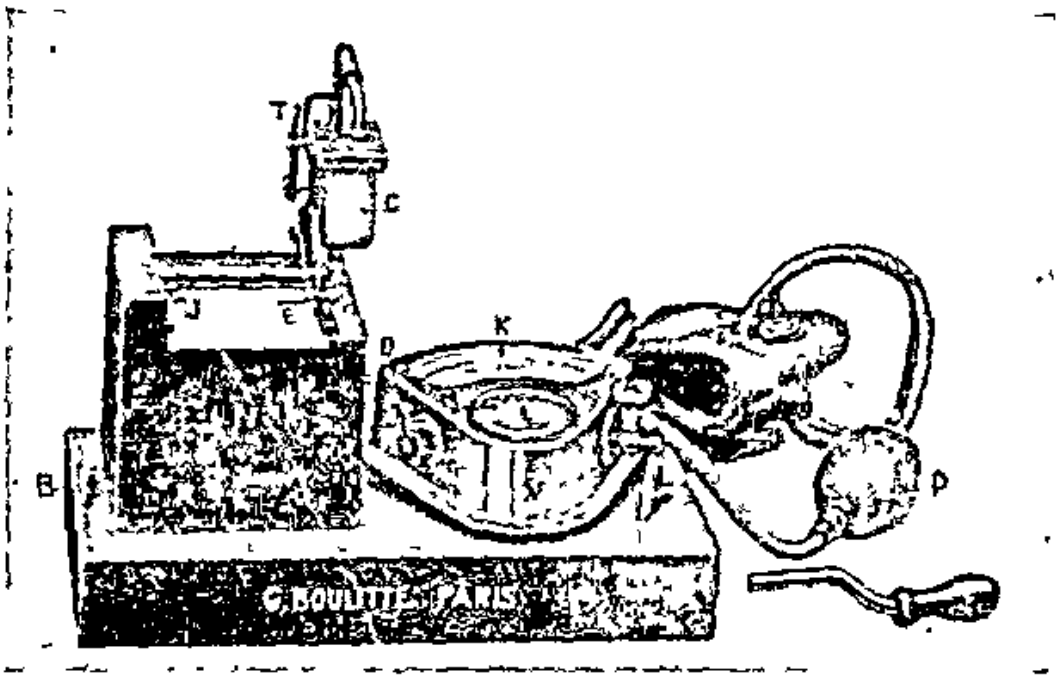


Fig 30.—Boulitte's portable recording oscillometer

which permits estimation of the maximal pressure (Fig. 32). If it is desired to save time, the three arterial pressures may be recorded on the same tracing by setting the distributor (D) first at Mx, and next as soon as this pressure is recorded at Mn, when the mean pressure (My on the tracings) is also registered.

3. Boulitte's Arterial Oscillograph.—This is a more elaborate and non-portable form of instrument, mounted on a light table with rubber wheels for use in nursing home or hospital. It yields a continuous and clearer tracing of the oscillometric curve.

4. The Tycos Recording Sphygmotonograph (Fig. 33).—This is useful for routine record of diastolic and systolic

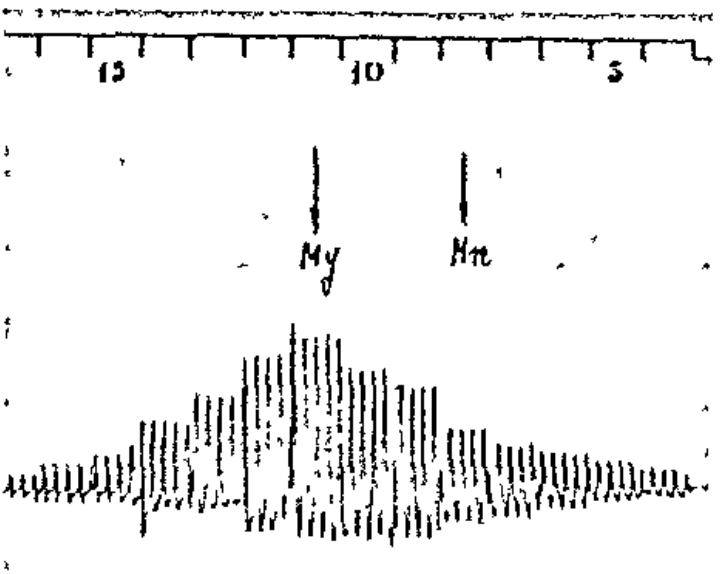


FIG. 31.

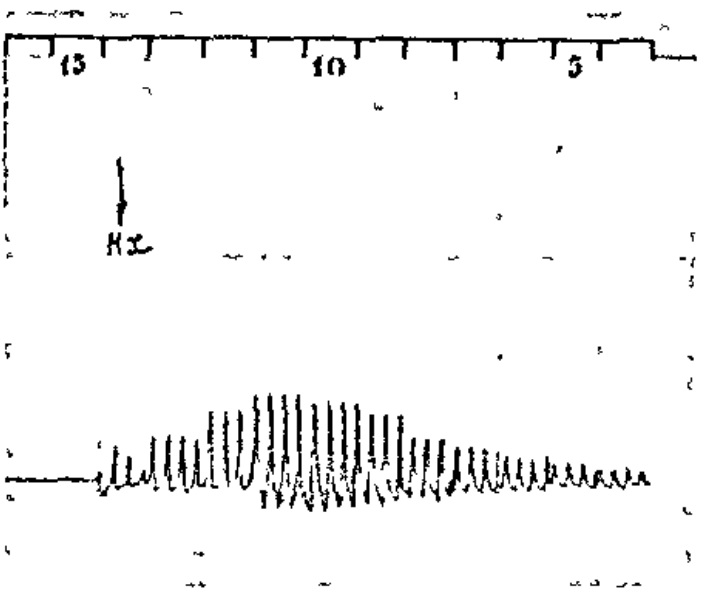


FIG. 32.

Figs. 31 and 32.—Tracings taken on millimetre-squared paper with Boulitte's portable registering oscillometer. The scale at the top registers in centimetres of mercury. Mn = diastolic pressure and My = mean pressure, obtained by adjusting a distributor valve so as to utilise both pockets of the armlet; Mx = systolic pressure, obtained by putting into action only the upper air pocket of the armlet.

pressures, which appear on the tracings with a fair degree of clarity, but since there is a damping down of each pulsation the upper portion of the successive pulse waves appear as straight lines, thus altering their essential characters

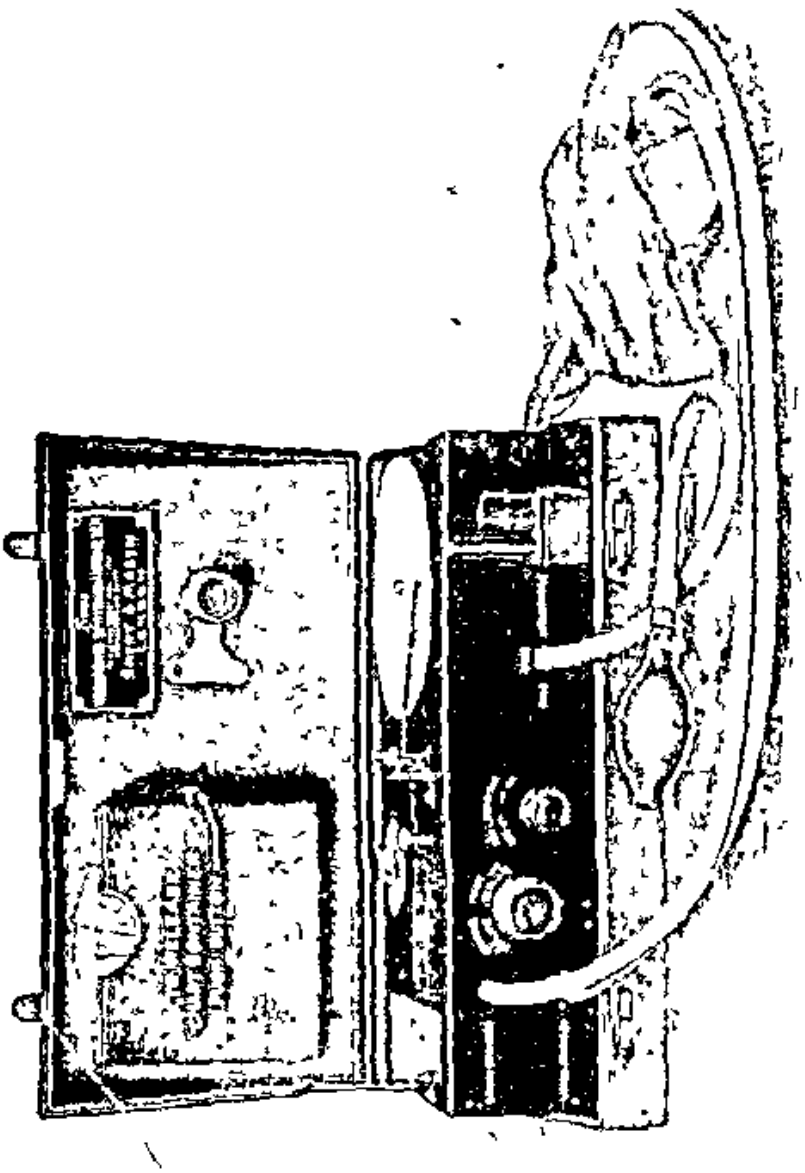


Fig. 33.—The Tycos recording sphygmotonograph. (By courtesy of Messrs. Short & Mason, Ltd.)

(Fig. 34). Moreover, the mean pressure cannot in most instances be determined.

Unless compensated by a device such as that of Gallavardin's double armlet, or by an internal adjustment of the apparatus itself, the graphic method of recording arterial pressure, as in Plesch's tonoscillograph, yields a systolic

reading several millimetres higher than afforded by the auditory method. To avoid confusion, the Tycos sphygmotonograph is compensated by adjusting the width of the compressing armlet so that the reading on the chart is the same as that obtained by the auditory method.

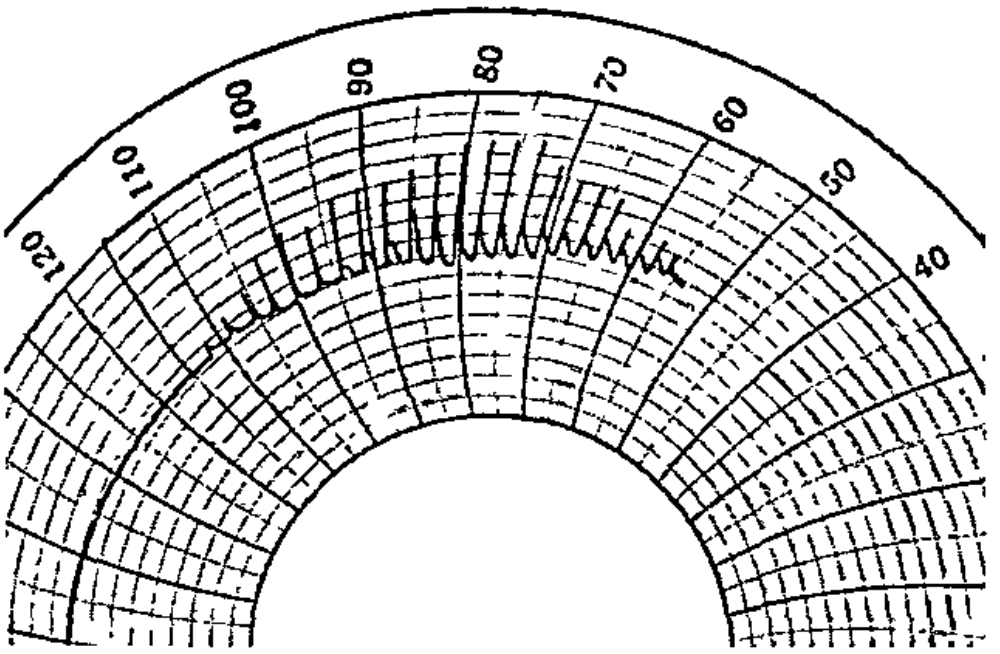


FIG. 34.—Sphygmotonogram of normal arterial pressure. Systolic = 111. Diastolic = 70. (By courtesy of Messrs. Short & Mason, Ltd.)

II. Direct Experimental.

The Boulittograph (Fig. 35).—This latest invention is comparable with the electrocardiograph in that by means of an optical arrangement a ray of light from an electric lamp is projected through lenses on to a roll of photographic paper travelling at a known rate of speed. A time marker is interposed so that the distance between two vertical lines on the paper corresponds to $\frac{1}{25}$ of a second, the speed being either 50 or 25 mm. per second. The horizontal lines indicate absolute pressure valves, each measuring 1 cm. of mercury (Fig. 36).

The *modus operandi* is as follows: the required limb is bared over the artery (femoral for choice, brachial, radial, etc.) to be investigated. A fine sharp needle with flexible lead tube attached is filled with an anticoagulant solution

and the artery is punctured. While the needle is in the lumen of the artery the oscillations due to intra-arterial pressure are observed, as the registering photographic band

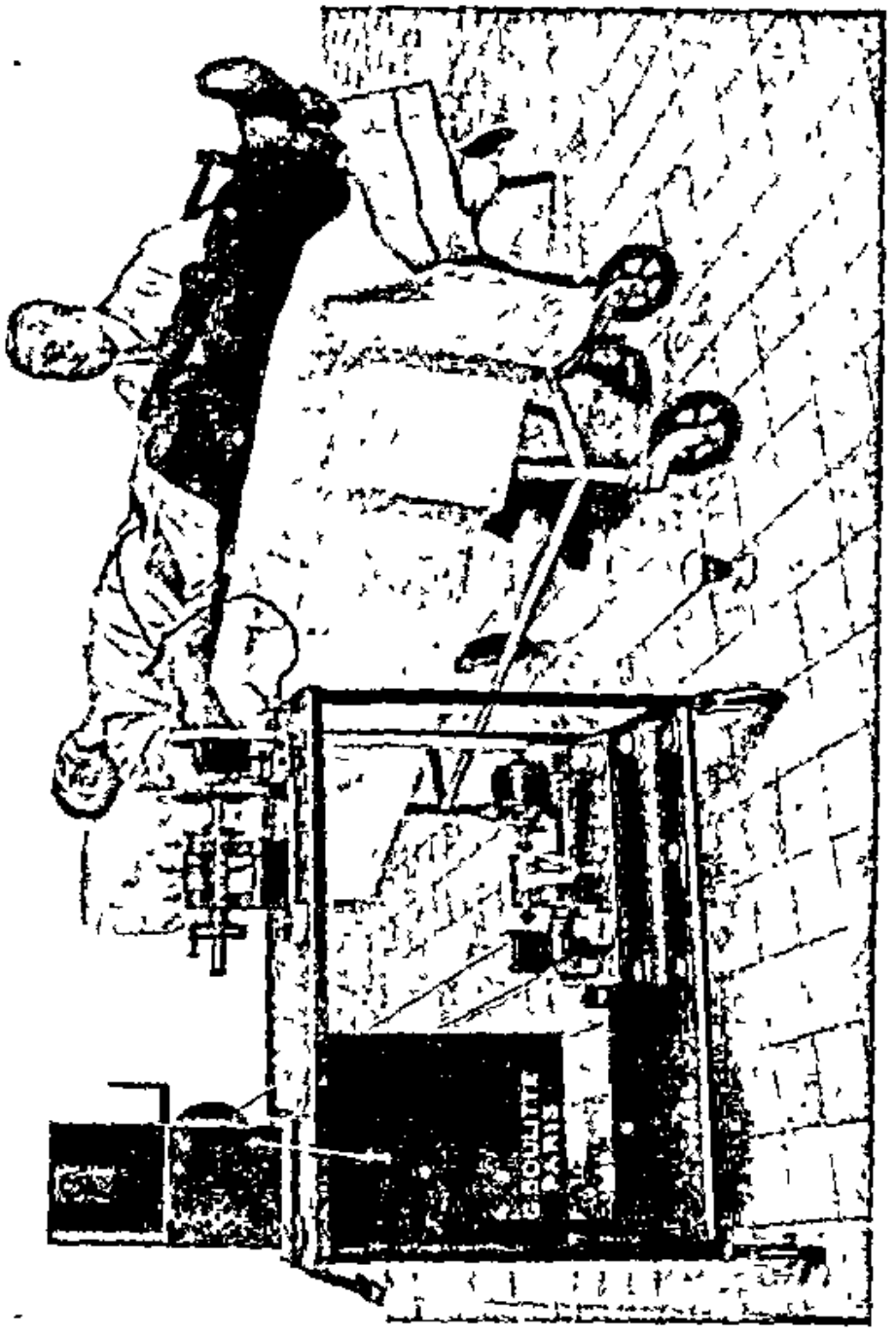


FIG. 35.—The Boulittograph.

of paper is allowed to unrol, through intervention of an index attached to a manometer connected with the blood-containing capsule.

In this way the actual intra-arterial pressures are recorded

for all their values during a series of cardiac cycles for as long a time continuously as the operator desires.

While of considerable interest to a physiological laboratory

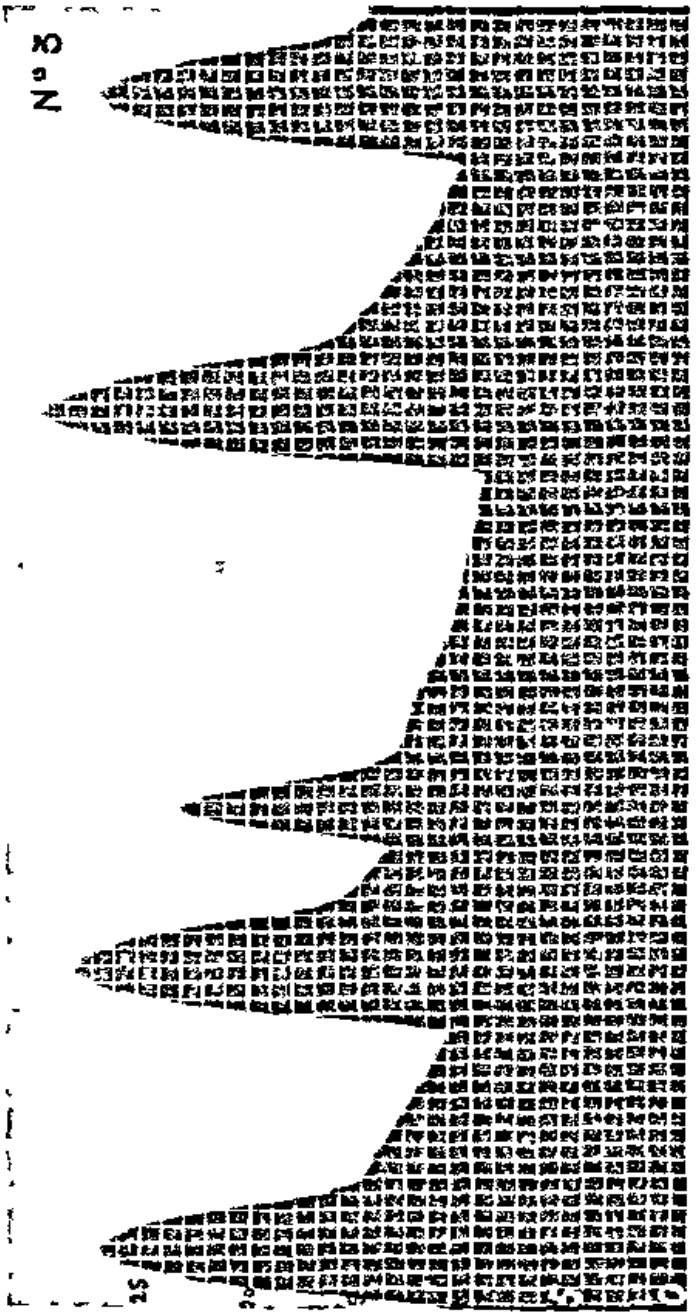


FIG. 38.—Record from femoral artery with the Boulitograph. Hyperpnœa with left ventricular enlargement. Speed 50 mm per second.

or hospital research unit, this method of direct puncture of one of the larger arteries would probably make considerably less appeal to the majority of private patients.

Blood Pressure Instruments to be Avoided

(A) *Mercurial.*—(*a*) All the older forms of instrument in which the mercury is apt to be spilt.

(*b*) Where the scale is not a true measure of the mercury displacement.

(*c*) Where friction results from oxidation of the mercury on the walls of narrow-calibre glass tubes, resulting in air-pockets.

(*d*) Where the mercury column shows considerable oscillation.

(*e*) Where the long arm of a U-tube has a defective joint.

(*f*) Where metal clips obscure some of the scale divisions.

(*g*) Where there are movable parts and mechanical devices subject to wear and failure of adjustment.

(*h*) Where the apparatus and box are too heavy or bulky for ease in transport.

(B) *Aneroid.*—(*a*) Where the mechanism depends on a spring diaphragm likely soon to get out of order or to wear out, which causes faulty position of the needle. The chief disability from which most aneroids suffer is that alternate expansion and contraction weakens their elasticity till no dependence can be placed upon the reaction of the diaphragm, which tends to undergo permanent distortion when frequently used for the determination of high pressures. Such distortion is usually indicated by failure of the needle to return to the zero point.

(*b*) Where the mechanism is difficult to repair.

(*c*) Where the needle moves in a jerky manner by reason of friction.

(*d*) Where there is an adjustable dial which may allow the zero point to coincide with a wrong position of the needle. The greatest errors are due to this cause and to the aneroid being tested at only two or three points on the scale, on the assumption that intermediate graduations should be uniform.

(*e*) Where a stop is fixed to the dial so that the needle always registers zero under atmospheric pressure alone.

(*f*) Where the apparatus is cheaply constructed with dial markings stamped by machinery and not individually

marked by calibration against a standard mercury manometer.

(*g*) Where the release valve is of trigger pattern, permitting a sudden excessive lowering of air pressure instead of a gradual one.

Other factors, not inherent in any particular make of instrument, which cause inaccurate readings are :—

(*a*) Where the instrument is subjected to variable temperature and barometric conditions, which induce contraction and expansion of the metal.

(*b*) Where the delicate adjustments become shaken or jarred as the result of a blow or fall.

(*c*) Where dust and grit work in to the bearings and cause faulty movements.

(*d*) Where the compression cuff is less than standard width. Too narrow a cuff may import an error reaching to + 40 per cent.

CHAPTER IV

Fundamental Physiological and Physical Factors in Blood Pressure

"A man's life may be said to be a gift of his blood pressure, just as Egypt is a gift of the Nile" Osler.

The physiological aspects of blood pressure are wide and far-reaching, necessitating, as they should, deep and intensive investigation of correlated physical and psychical phenomena.

It is, however, far beyond the scope of this manual to enter into a detailed consideration of all the principles and data involved I will, therefore, limit myself to a synopsis of the fundamental factors concerned in arterial pressure, which will serve as an introduction to its clinical study

In this chapter I propose briefly to discuss the leading physiological and physical factors on which clinical variations of pressure depend, and from this groundwork to trace in succeeding chapters the origins of departures from standard levels which constitute either physiologically or pathologically high or low arterial pressures

Significance of Blood Pressure

First of all, what meaning should the term "blood pressure" convey ?

(*a*) *In the physical sense* blood pressure may be defined as that pressure which the blood exerts at a given instant upon a given point in the circulatory system.

(*b*) *In the physiological sense* the term includes pressures which may be intra-auricular, intra-ventricular, arterial, capillary or venous

Arterial blood pressure is a force *originated* by ventricular contraction, *maintained* by the reaction to distension (resiliency) of the arterial walls, and *regulated* by the degree of resistance in the terminal portion of the arterial system.[16]

The five main factors concerned in its variations are two fundamental and three subsidiary factors, namely :—

Fundamental Factors

1. The energy of the heart, as measured by unit output (an intermittent force).
2. The peripheral resistance (a continuous force).

Subsidiary Factors

3. The resiliency of the arterial walls.
4. The volume of the circulating blood.
5. The viscosity of the blood.

In physics there are only two sources of rise of pressure, **increased force or increased load.** From the physical standpoint the human circulatory system consists of a **central**

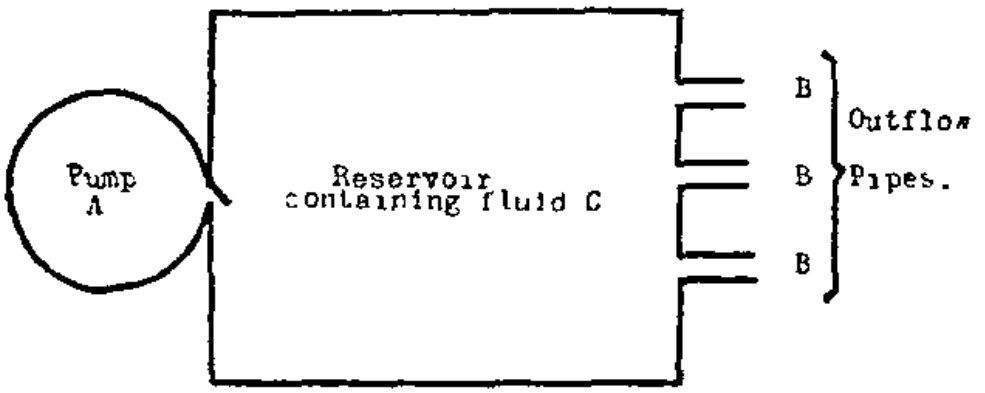


Fig. 37.—Schema of pressure system (diagrammatic).

pump system, an intermediate reservoir, and a peripheral outflow system comprising the series of pipes through which the circulating fluid is distributed throughout the body. In other words, an intermittently acting pump (heart) forces a liquid (blood) into a system of large elastic tubes (aorta and its branches, which act as a second heart), which divide (smaller arteries) and sub-divide (arterioles) to a terminal network (capillaries), from which the liquid is returned through another set of tubes (veins) to the inlet of the pump.

Pressure is varied by the force of the pump A (Fig. 37), the size of the distributing vessels B, and the character of the

circulating fluid C. Variations in the pressure can only be produced by an alteration in one or more of these conditions.

1. If the force of the pump A be increased, and B and C remain the same, the pressure is augmented. If the force of the pump be decreased, the pressure is diminished.

2. If the force of the pump A remains the same, and C is unchanged, then, if the diameter of the pipes B is reduced so as to obstruct the outflow, the pressure is again raised, and if the diameter of B be increased, the pressure falls. In physical parlance this peripheral resistance is termed the load. Hence, if C remains constant, *i.e.*, if the condition of the circulating fluid remains the same, a rise of pressure must be caused either by an increase of the central pump force or by an increase of the peripheral resistance.

The central system may be affected in two ways: primarily by direct increase of pump force; or, secondarily, increase in peripheral resistance may demand increased action on the part of the pump, so that the increased resistance may be overcome by an augmented central pressure.

The peripheral resistance, or load, may also be affected in two ways: primarily, when there is a direct alteration in the size of the peripheral vessels, or a change in their resiliency in the direction of increased rigidity; and, secondarily, where these alterations take place in order to compensate the augmented dynamic action of the central pump which necessitates contraction or rigidity of the peripheral vessels.

Hence the primary causes of increased pressure are only two in number: either (1) a central dynamic cause due to increased power of the heart and great vessels, or (2) a peripheral load increase due to increased pressure, vascular thickening or rigidity of the vessels. Any secondary cause is only compensatory to the primary. An expression of these physical terms in equivalent terminology will be found in Table VII, p. 102.

It should be borne in mind that the greater part of the cardiac energy is expended, not in driving the blood through the vessels, but in distending the walls of the arteries.

The more efficient is the circulatory mechanism, the lower is the pressure required to circulate a given volume of blood.

If the individual cardiac contraction becomes quicker and more powerful, though the volume of blood put out with each beat is unchanged, the systolic pressure will rise and the diastolic remain stationary; if the peripheral arteries contract, and their resistance to blood flow becomes increased, both systolic and diastolic pressures will be elevated. A drop in the systolic and diastolic pressures will occur if each heart-beat becomes prolonged or less forceful and if the arterioles relax. In blood pressure changes with rest or emotional stimuli both these factors are concerned.[57]

The systemic arterial pressure far exceeds that in the arteries of the pulmonary circuit. The reason is that a highly variable physiological resistance exists in the systemic arterioles, whereas in the pulmonary circuit no such resistance occurs, since in the lungs there is no necessity for one inflated portion to receive more blood from the pulmonary artery than another, for all portions fulfil the same function, *i.e.*, aeration of the blood. When an organ demands an increased blood supply, all that is needed is for its arterial system (notably the arterioles) to dilate, when the blood is driven under a high pressure through the corresponding capillaries. These vessels are capable also of active contraction and dilatation, these phases probably coinciding actively with corresponding arteriolar phases in response to the tissue needs of the moment.

In the clinical sense "blood pressure" should be held to include diastolic, mean and systolic pressures.

The common acceptation of the term as denoting only systolic arterial pressure, which when stated as a number —*e.g.*, 160—is usually taken to denote "systolic pressure in the brachial artery at the time of investigation measured in millimetres of mercury," is too narrow, since it represents only a portion of the whole which it should portray. At each estimation the observer should be satisfied with nothing less than a record of the systolic pressure, the diastolic pressure, the differential pressure, the pulse rate and the figures for the latter two multiplied together. These five essentials together constitute what I have termed "the complete arterial pressure picture" (p. 27). If oscillometric methods be used, the mean pressure can also be recorded.

Minimal (Diastolic), Maximal (Systolic) and Mean Arterial Pressures

Because the driving force of the left ventricle is not continuous, but intermittent and pump-like, the output

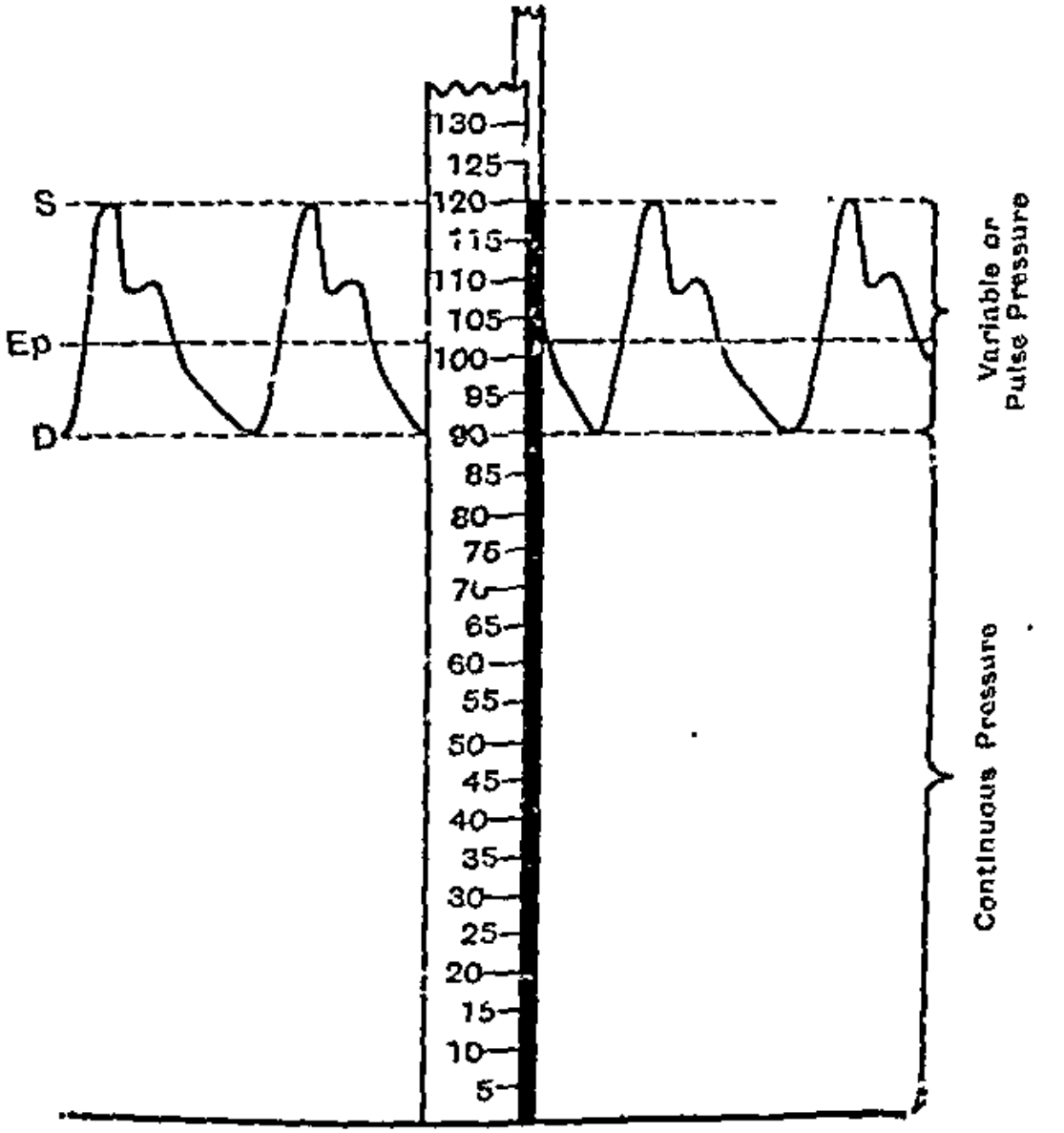


FIG 38.—Diagram to illustrate the continuous pressure which the arteries have to sustain below the diastolic level D and the superadded pressure which occurs during systole. The variable or pulse pressure, in this example 30 mm. Hg, is the difference between the systolic level S at 120 mm and the diastolic level D at 90 mm. Ep = level of mean pressure ("efficacious pressure").

of blood is sent forward in waves, which rhythmically distend the walls of the arteries and so produce the pulse, the base of each wave corresponding with the least, or

minimal, pressure, whilst the crest of each wave corresponds with the greatest, or maximal, pressure (Fig. 38).

Since the minimal pressure occurs between successive ventricular contractions, *i.e.*, during the resting time of the heart (diastole), such pressure is also termed "diastolic," whilst to the maximal pressure the term "systolic" is applied, because it happens during ventricular systole, *i.e.*, that portion of the cardiac cycle during which the ventricles are in process of contraction.

Hence it is incorrect to speak of *arterial blood pressure* as a fixed and definite entity for any individual. In reality it is an amount which *is constantly fluctuating between certain minimum and maximum values characteristic for each subject under like conditions of observation.* At the outset, therefore, it is of the greatest importance to remember that, of the two pressures, **the minimal pressure** is the more valuable in that **it is a measure at the moment of a variable burden which,** during the life of the individual, **the arteries and aortic valves must continuously bear, and from which there is no escape, whilst the maximal pressure indicates only an intermittent and superadded load.**

Differential (Pulse) Pressure.—Differential pressure is the difference between the diastolic and systolic pressures; *e.g.*, a diastolic pressure of 86 and a systolic pressure of 130 will yield a differential or so-called "pulse" pressure of 44.

Mean Pressure.—In laboratory experiments on animals the mean pressure has long been recorded, but in clinical medicine no importance was attached to it until the observations of Pachon in 1921. Since that time a series of publications by the French school of medicine, notably by Astier, Fontan, Dodel, R. Roger, Chevallereau, Escaich, Vaquez, Kisthinios, Gley and Gomez,[52] have assigned to this measurement a value which can only be regarded as somewhat artificial in comparison with the real and vital value of the diastolic pressure. The mean is not, as commonly supposed, the arithmetical mean between the diastolic and systolic pressures, but is the average pressure at a given point which, in a pulse tracing, appears at a level often, but not necessarily, nearer the diastolic than the systolic pressure. The shape of the curve being, however, very variable, there

is no fixed or absolute relationship between the mean pressure and the minimal and maximal extremes. The above points are clearly shown in Fig. 37.

In practice, **the mean pressure is that pressure within the armlet which corresponds with the oscillometric index, that is to say, the largest oscillation measured during the course of an oscillometric investigation.** This largest oscillation follows immediately upon complete decompression and elastic distension of a previously constricted artery under the influence of a wave of pressure corresponding with cardiac systole. Hence **two basic factors enter into its composition, (*a*) the force of the cardiac impulse, (*b*) the degree of arterial constriction or relaxation in the vessels under investigation.** Both these factors are variables, so that unless one is known, or remains constant during observation, the other cannot be inferred. Thus, although variations of the index in the same subject is helpful in symptomatology, a comparison of respective values cannot directly be made in different subjects.

Under varying mechanical, chemical or physical conditions the same mean pressure will not yield the same circulatory output. Hence consideration *solely* of this pressure does not suffice in assessment of circulatory efficiency or inefficiency. This conclusion has been reached by Pachon and Fabre [58] and confirmed by Lian.[59]

From the foregoing it would appear that no solid grounds exist for attributing to the mean pressure the "great importance" which by the French School, under the influence of Pachon and Vaquez, it is stated to possess. All arterial pressures are "dynamic," hence the adjective should not be limited solely to the mean pressure, nor are the terms "efficient" or "efficacious" any the more applicable in view of the conclusions mentioned in the preceding paragraph.

The Aim of Blood Pressure.—The aim of blood pressure is the maintenance of blood flow, and the strong elastic recoil after distension of the great aortic reservoir is one of the leading factors by which an efficient circulation is sustained, since in recoil the aortic reservoir functions like a second heart.[16]

The Peripheral Resistance.—The peripheral resistance is a combination of several elements, including viscosity of the blood and variation in calibre of the smaller arterioles and capillaries under the influence of the vasomotor system and the external pressure exerted upon them by the tissues.

The Sympathetic Vasomotor Nervous System.—This system consists of a vasomotor centre in the medulla, with subsidiary centres in the spinal cord. The paths for vasomotor reflexes are receptor (afferent) and excitor (efferent). The receptor paths are (i.) pressor and (ii.) depressor. "All sensory nerves are pressor in their action, causing the vasomotor centre to throw out increased constrictor impulses, particularly to the splanchnic area. This tends to produce a rise of pressure. . . . The only pure depressor nerve is the depressor branch of the vagus. It provides a way of escape for the heart when labouring against too high a blood pressure. But depressor fibres may also be demonstrated in sensory nerves."[60] Stimulation of these brings about a fall of pressure. The excitor channels are also of two kinds: (i.) constrictor and (ii.) dilator. Of these the vasoconstrictor are by far the more numerous and important in controlling blood pressure.

The vasomotor system as a whole is concerned with the regulation both of general blood pressure and local blood supply, and, as we have already seen (pp. 13, 15), when stimulated by the pressure of the armlet modifies arterial tonus as expressed in the vibrant and sonorous qualities of the arterial wall. As Barbier[17] has well said, "le sympathique est le tendeur du tambour artériel."

Tone, Tension and Viscosity

Not infrequently confusion is apt to arise as a result of lax use of the terms "tone," "tension," and "viscosity," and since each of these has a distinct bearing on the subject, it is necessary to outline their several significations.

Tone.—During life the arteries, large and small, may be much constricted as a result of contraction, or much dilated as a result of relaxation of their muscular fibres; or, again, during long periods they may be kept in a state

of moderate contraction (tonus). Such variations in calibre are brought about by activity of the vasomotor nervous system, the first and third states being caused by stimulation of vasoconstrictor fibres, the effects being both local and general, whilst the second occurs from stimulation of vasodilator fibres, the effects being limited to special vascular areas, in response to local requirements of organs or tissues.

Tonus (or tone) is thus an active state of the smooth muscle of the arterial wall which determines and regulates its kinetic function. Muscle tone depends on muscle consistency, which latter has to do with stratification of the muscle cells. With increased tonicity the vessel becomes harder, and the difference is appreciable to the touch. "Tone in a vessel is that which preserves its mean diameter, the due proportion between the extremes of dilatation and recoil, and has furthermore the somewhat different virtue of keeping the vessel wall well home upon its contents." [61]

Arterial tone as a component of blood pressure cannot be ignored, but at times its importance is apt to be magnified, and in assessing the quality of the pulse, as I have already said, the finger of itself is often insufficient, since the calibre of any artery can undergo considerable alterations.

Tension.—"Tension" is frequently used, especially by the French and American schools, as synonymous with "pressure," *e.g.*, "pulse tension," "arterial hypertension." Tension, however, implies a *pull*, while pressure implies a *push*. In any event, "supertension" should be read for "hypertension." With reference to blood pressure, however, it is more correct and less obscure to drop the latter hybrid term altogether. "To the coats of an artery only can the word 'tension' apply. The 'blood' cannot be tense in any but an abstruse mathematical sense. . . . Tension is the stress which tends to split the artery longitudinally or transversely, and such stress is at more advantage when the vessel is relaxed." [62] In his last publication Allbutt observed that "hypertension" (or, to speak more grammatically, hyper*tonus* or *super*tension) denotes too narrow a meaning: "tension" can be predicated only of

the proximal distended vessels, and in them is not the essential factor; the essential factor is the rise of blood pressure." [63] The effects of tension are best seen in an advanced case of aortic regurgitation, but "hypertension," as applied to a blood pressure which is raised, can only be regarded as a misused term

Density, Specific Gravity, Viscosity.—Density is the proportion of mass to volume or bulk. Specific gravity is the weight of any given substance as compared with the weight of an equal volume or bulk of water or other standard substance at the same temperature and pressure, and is a function of density. Viscosity is the property of a liquid whereby it resists the relative motion of its parts. This property is analogous to that of friction. For example, water flows easily on itself or on other surfaces, whereas treacle or some of the heavier oils do so with greater difficulty. On the other hand, the specific gravity of water is often higher than that of a treacly oil.

Viscosity, therefore, is *not* specific gravity, for high specific gravity may co-exist with low viscosity. Viscosity connotes increased resistance to the flow of a liquid, so that, if we accept Allbutt's view that "nearly 200 times more of the heart's energy is expended in overcoming friction than in direct transference of velocity to the stream," "we shall scrutinise most carefully any degrees, however small, in the stickiness of the circulating fluid." [60]

Blood viscosity depends on two main elements :—

(*a*) Colloid, the degree of viscosity varying directly with the size of the individual colloid molecules contained in the liquid, the albumin molecule being larger than the globulin molecule. Thus an increased ratio of albumin to globulin points to an increased blood viscosity.

(*b*) Crystalloid. Blood viscosity is also capable of being increased by a preponderant content of heavy metallic ions, such as ionised calcium.

Blood density curves closely agree with those for percentage of hæmoglobin and number of erythrocytes. Hence in true splenomegalic polycythæmia (erythræmia) the blood viscosity, as measured outside the body, is greatly increased. In spite of this, however, arterial pressure ranges

only slightly, if at all, above the average, and the left ventricle is not usually hypertrophied. Variations in density which are readily balanced by increase or decrease of water content absorbed from the tissues do not largely affect arterial pressure. So that we may conclude that, although density has its share in maintenance of blood pressure, yet changes in consistence and viscosity can readily be balanced by the tissues.

Investigations now proceeding all over the world into the biochemical composition of the blood and further elucidation of colloid and crystalloid phenomena which take place therein may possibly afford assistance in solving the problem of the causation of high arterial pressures, and in particular of the diastolic pressure, although up to now no direct relationship between blood viscosity and blood pressure has been established.

CHAPTER V

Physiological Variations in Arterial Pressure

"We are apt to conceive too uniform a notion of blood pressures, to regard them as moving with a piston-like action on reciprocating planes; whereas a better comparison would be with the waves of the sea, or with the wafting undulation of a large bird."

Allbutt: *System of Medicine.*

Once the fundamental factors (outlined in Chapter IV.) on which blood pressure depends are clearly grasped, the reader will find little difficulty in tracing the processes of physiological variation, and, from a comprehension of these, can logically follow the sequence of events which occur in disordered and diseased states.

From the way in which "blood pressure" is often spoken of, it would appear that a certain blood pressure is assumed by many to be as characteristic of an individual as are his physical peculiarities. But this is not so, for though usually confined within certain general limits, yet such pressure is not constant from month to month or from day to day, but, from respiratory, psychical or other causes, may fluctuate widely and rapidly both in health and in disease, even during the brief time of investigation. Hence the maximal and minimal bounds between which arterial pressure normally rises and falls are the important criteria which in every case we must definitely determine.[64]

Significance and Importance of the Diastolic Pressure

Since the methods by which diastolic pressure can readily and accurately be determined have been described only within recent times, and because earlier workers had perforce to be content with registering systolic pressures alone, it is, perhaps, not altogether surprising, though most

unfortunate, that even in certain modern books the authors have contented themselves with recording only systolic pressures, thus hampering greatly the usefulness of their otherwise admirable work.

A few there are who still think that registration of the diastolic pressure is an irksome and needless refinement, and that for all practical purposes a record of the systolic pressure suffices.

To record the maximum pressure whilst ignoring the minimum not only affords no indication of what the mean pressure is likely to be, but resembles attempting to solve a complicated problem of which only one factor is given. "No fact regarding the blood pressure is better established than its wide range of variation in any individual. It is, therefore, impossible to speak of a normal value for blood pressure, but only of certain normal upper and lower limits."[65]

In short, records of systolic pressure alone are of comparatively little value, because the knowledge which such limited data afford is vastly inferior to that which is obtained from a consideration of the complete arterial pressure picture of systolic pressure in relation to diastolic and pulse pressures and pulse rate

The reasons which cause me to reaffirm that the diastolic pressure is of paramount importance are as follow :—

1. Diastolic pressure is the measure both of peripheral resistance and of vasomotor nervous tone. Since the degree of peripheral resistance can thus be inferred, the diastolic pressure may be considered to be a better index of high arterial pressure than is the systolic pressure.

2. In relation to systolic pressure the diastolic pressure is the more important in that it is Marey's[66] "constant" element in arterial pressure, transitory systolic elevations which form the pulse representing only an intermittent and superadded load.

3. The diastolic pressure is the measure of the load which throughout life the arterial walls have continuously to support (Fig. 38), and is significant not only from the effect produced in them by mechanical tension but "with regard

to the influence of an excessively high diastolic pressure upon their blood supply, especially in the vessels of the lower extremity, where, in the erect posture, the diastolic pressure is greatly augmented by the influence of gravity." [18]

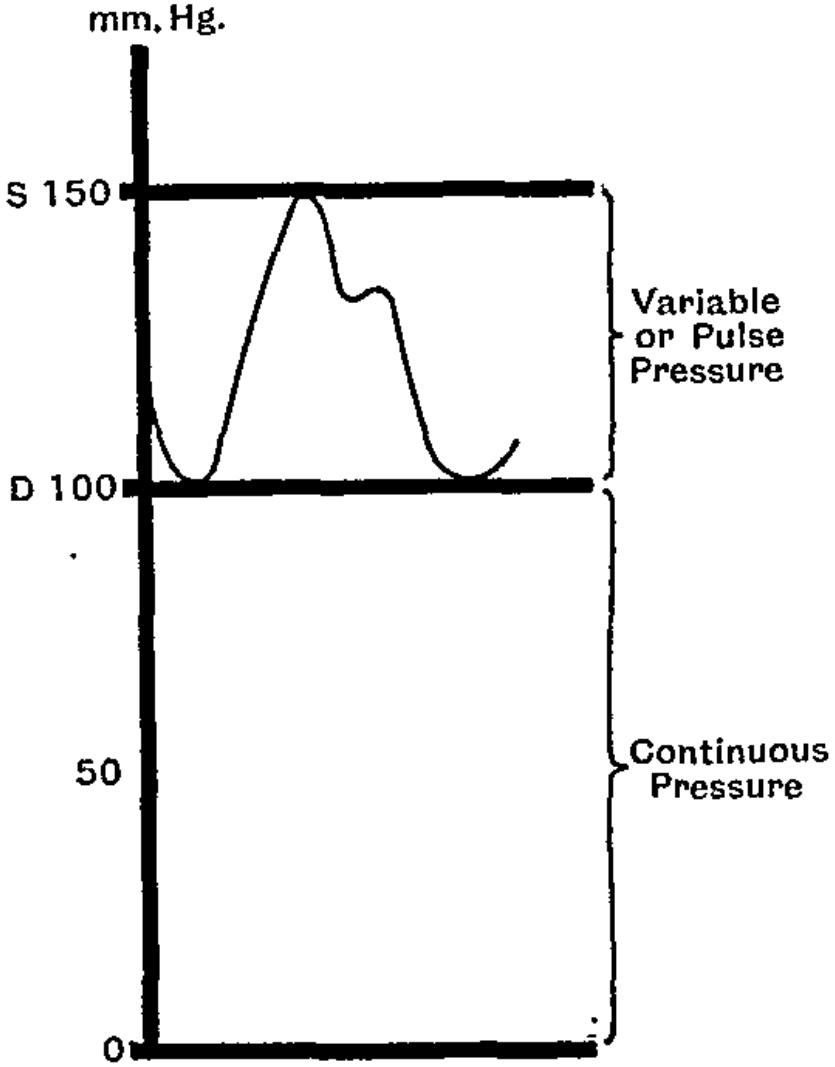


FIG. 39.—Diagram showing the relation of the diastolic to the systolic pressure. In this example the figures for the respective pressures are arbitrary.

4. The diastolic pressure is the measure of resistance in the aorta which has to be overcome by the blood stream in separating the aortic valves during the initial stages of left ventricular contraction (Fig. 40).

5. It is one of the indices of the driving force of the heart and of the eliminative capacity of the body.

6. In relation to systolic pressure it constitutes the additional necessary factor in the equation for determining the differential pressure (*Fig.* 41).

7. The systolic, diastolic and differential pressures, when conjoined with the pulse rate, and the product of the figures for the two latter together make up "the complete arterial pressure picture."

8. The diastolic pressure is *increased* by causes which produce vasoconstriction and so heighten the peripheral resistance, *e.g.*, aortic stenosis or lowered temperature; at times by increased rapidity of cardiac action; by increased intracranial tension, *e.g.*, cerebral tumour, meningitis, and in certain cases after exercise.

9. Gradual increase of diastolic pressure means harder

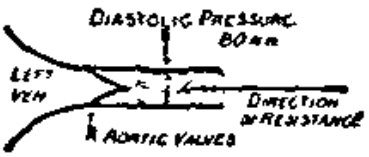


FIG. 40.—Diagram showing the left ventricle and arterial system during diastole. The diastolic pressure is 80 mm. and represents a resistance borne by the aortic valves and arterial walls and directed contrary to cardiac force. The power furnished by the heart must be sufficient to equalise this resistance and enough in excess to render its work potential in the arterial wall.

work for the heart to supply the parts of the body with blood. As compared with the transient systolic elevation at each heart beat, the significance of the diastolic pressure in relation to bursting of a degenerated cerebral artery is great. "This is illustrated by the comparative rarity of cerebral hæmorrhage in cases of aortic regurgitation, with its high systolic but low diastolic pressures."[67] In arteriosclerosis, increased diastolic pressure is accompanied by increased differential pressure and increased size of the left ventricle temporarily (exercise) or permanently.[18]

10. The diastolic pressure is *lowered* by causes which produce vasodilatation, and so lower the peripheral resistance, *e.g.*, fevers, certain cases of hyperthyroidism and neurasthenia, sometimes by conditions which slow the heart rate, *e.g.*, heart block; by diminished resiliency of the

arterial walls in the absence of increased peripheral resistance; and by aortic insufficiency.

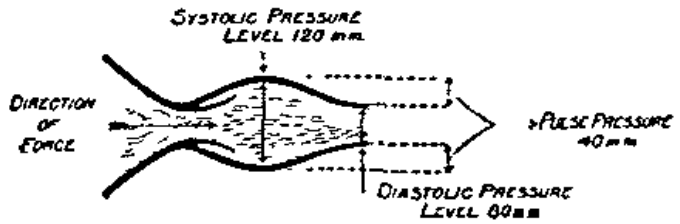


Fig. 41.—Diagram showing left ventricle and arterial system during systole. The diastolic resistance (peripheral resistance) of 80 mm has been overcome by a systolic force of 120 mm The difference between these two pressures, the pulse pressure, is 40 mm., and represents the efficient work of the heart in maintaining the circulation (Figs. 40 and 41 by courtesy of Messrs Short & Mason, Ltd)

Significance of the Systolic Pressure

Systolic pressure indicates the maximum cardiac energy at a given moment, together with the degree of peripheral resistance. It fluctuates within wide physiological limits in response to the various activities and needs of the body, being modified under conditions of sleep, rest, posture, food, exercise, emotion, etc. (pp. 94 *et seq.*), the intermittent and more or less regular transitory elevations of systolic pressure above the more stable diastolic level constituting the pulse wave.

Significance of the Differential (Pulse) Pressure

Differential or pulse pressure is not a direct measure, like the minimal and maximal pressures, but only the difference between these two variables, upon which its attributes depend.

Clinically, the differential pressure represents the load of the heart. Systolic and differential pressures yield myocardial values, whilst diastolic pressure indicates the degree of arterial and arteriolar resistance.

Wide variations in the differential pressure from standard readings at various ages are possible, the coefficient of variation for differential pressures being more than twice as large as the systolic in some age periods, and nearly twice as

large as the diastolic coefficients. Standard averages remain about 44 mm. between ages twenty-five and fifty-five, after which they show considerable increase because of the faster rise in the systolic than in the diastolic pressure.

In adult life the normal range of differential pressure is from 30 to 55 mm. Expressed otherwise, under conditions of normal pressure balance differential pressure is about 40 per cent. of the average systolic reading and 65 per cent. of the average diastolic. A differential pressure reading below 30 mm. suggests an error of observation, except in a young child.

The higher the maximal pressure the larger usually is the differential pressure. Differential pressure also depends on pulse rate. With a slow pulse the blood stream has more time to flow through the arteries in diastole. Hence diastolic pressure will be lowered and differential pressure increased. In general, the slower the pulse rate the larger is the differential pressure; the more rapid the pulse rate the smaller is the differential pressure.

Significance of the Mean Pressure

In direct contrast to systolic pressure, mean pressure is a physiological constant in the same subject, the standard range being from 80 to 100 mm., with an average of 90 mm. Its physical equivalent is the oscillometric index (p. 76), which bears to the minimal pressure a ratio normally of about one-third. This ratio is linked with the heart rate, decreasing with increased systolic effort and with increased peripheral resistance. Any figure for mean pressure exceeding 110 mm. Hg. may be regarded as pathological. At times the onset of hyperpiesis is signalised by rise of the mean pressure alone. Wide variations of extremes of pressure are not dangerous provided mean pressure remains relatively stable, whereas variations of mean pressure induce grave circulatory disturbances.[68]

Average Normal Arterial Pressures

I. In Children.—Arterial pressures gradually rise from birth, at which the systolic pressure varies between 20 and

60 mm. Hg ; in infants up to two years it averages 80 mm. The diastolic pressure is relatively higher, and the differential pressure is about 10 mm. less than in adults.[69] The systolic pressure shows a slight but gradual rise from three to ten years, after which the increase is more abrupt, with a rapid elevation in the fourteenth year during adolescence.

The following table of average arterial pressures from ages three to fifteen is based on 2,300 observations on well-nourished schoolchildren of both sexes by Judson and Nicholson,[70] who employed the auscultatory method checked by a modification of Erlanger's instrument.

TABLE I

Average Arterial Pressures in Children

Age, years	Width of cuff in centi metres	No of observa tions	Systolic pressure, millimetres of Hg	Diastolic pressure in milli metres of Hg, at beginning of 4th phase	Differen tial (pulse) pressure	Pulse Rate	D P × P R
3	9	24	92 0	58·4	33 6	92	3,091
4	9	95	92 6	61·7	30·9	99	3,059
5	9	69	91·6	60 0	31·6	93	2,939
6	9	110	93·8	63·5	30·3	95	2,878
7	9	145	87·9	64 2	22·7	87	1,975
8	13	128	93 0	59·6	33·4	88	2,939
9	13	149	91·7	62·2	29·5	84	2,478
10	13	203	99·0	64·6	34·4	87	2,993
11	13	169	95·8	62·3	33·5	87	2,914
12	13	94	99·9	59·6	40·3	89	3,587
13	13	80	104·0	63·2	30·8	96	2,957
14	13	43	105·8	63·7	42·1	84	3,536
15	13	35	99·6	61·8	37·7	84	3,167

In boys and girls of from four to sixteen years of age, Faber and James [71] have stated that the mean systolic pressure shows no significant differences between the sexes, whereas *the mean diastolic and mean differential pressures* show significant sex differences. Standard deviations are

greater for girls, indicating a normally greater variability in females, especially during adolescence.

In British boys Stocks and Karn [72] find lower systolic averages at age 5 than those recorded by the two sets of American observers just quoted but a considerably higher level at age 14 (115 mm.)—a rise of 30 mm., nearly twice that found by the latter authors. Stocks and Karn find the accelerated rise between ages 13 and 17 to amount to 16 mm.

Melvin and Murray 36 found the auscultatory method easily applicable to twelve healthy children of ages between eight and fourteen, who gave an average systolic value of 107 mm. and a diastolic value of 74 mm., with an average pulse pressure of 33 mm.

"The rising pressure of childhood undergoes some acceleration about puberty and reaches the normal adult level somewhere in the age period of seventeen to twenty; there is some evidence of a slight lowering in the early adult years. The pressure then alters little up to the age of about forty, after which a more definite progressive rise becomes manifest." [67]

II. **In Adults.**—Both for children and adults reliable British figures are few. American figures both for healthy children and adults are lower than those usually found in this country. Whether the explanation lies in differences in environment, including climate and diet, or along other lines, is uncertain.

Readings by Continental and American observers under various conditions of observation place the level of the diastolic pressure at between 65 and 110 mm. for adults, which accords with the figures given by Janeway.[65] This upper limit, based on observations with the older types of instruments, is far too high, and it is in accord with later experience that a diastolic pressure persistently above 90 mm. is to be regarded with suspicion, whilst one of 95 mm. or over is definitely pathological.

All the following figures are stated on the assumption that the blood pressure is estimated in the absence of psychical and physical disturbing factors, the subject being seated with muscles relaxed. My own series of averages I have set out as follows :—

TABLE II

Standard Arterial Pressures for Adults at all Ages

—	Systolic Pressure.	Diastolic Pressure.	Differential Pressure.
Men . .	127	83	44
Women .	123	81	42

Further observations with modern instruments on standard normal arterial pressures at various ages, especially as to the diastolic level, are still required, since one can only place reliance on those which have been collected during the past few years for four chief reasons: (1) Pioneers of blood pressure investigation dealt only with small numbers, which are insufficient to obviate experimental errors due

TABLE III

Systolic Pressure—Men Only

Age.	Symonds.	Mackenzie.	Rogers and Hunter.	Average Totals.
15—19	121·2	119	120	120 06
20—24	123·4	122	122	122·5
25—29	123·9	123	123	123·3
30—34	123·8	124	124	123·9
35—39	124·9	126	125	125·3
40—44	126·5	127	127	126·8
45—49	128·4	129	129	128·8
50—54	130·9	132	133	131·9
55—59	133·9	135	134	134·3
60 and over	135·2	137	—	136·1
Entrants .	150,419	18,637	62,000	231,056 Total Entrants

to personal equation and to fulfil the law of averages. (2) All earlier records and, unfortunately, many later ones are concerned solely with systolic pressure. With old apparatus this deficiency is comprehensible, for diastolic pressure could not be registered with any accuracy, and therefore was so variable as to be of little or no importance. (3) Early types of armlet, which were all too narrow, necessarily caused readings to be too high. (4) All the American assurance figures are vitiated for clinical purposes, because of the arbitrary choice of the very end of the fourth phase, just before silence, as the criterion of diastolic pressure, a point which is always too low, and not infrequently far too low.

Notwithstanding this defect, which is in some cases serious, it is to American life insurance that we are indebted for the only data of arterial pressure which have to do with vast numbers of healthy subjects. Five comprehensive

TABLE IV

Average Systolic and Diastolic Pressures (Symonds)

Age.	Men.		Women.	
	Systolic.	Diastolic	Systolic.	Diastolic.
15—19	121·2	77·7	119 2	77 2
20—24	123·4	79 6	120 6	78 2
25—29	123·9	80 5	120·9	78·8
30—34	123·8	81·5	121·7	80·3
35—39	124·9	82·7	123·3	81·8
40—44	126·5	83·8	126 0	83·4
45—49	128·4	84·9	128·2	85 0
50—54	130·9	86 3	130 9	87·1
55—59	133·9	87·0	134 8	87·8
60 and over	135·2	86·6	135 5	89·8
Entrants .	150.419	60.733	11.937	5,276

reports are to hand, namely, those of Fisher (1914),[73] Mackenzie (1915),[74] Rogers and Hunter (1919),[75] Goepp (1919),[76] and Symonds (1923).[35]

Table III. has been compiled from the records of three series of observations taken under similar conditions and age groups.

Fisher deals with an additional 19,339 cases, the average systolic pressure for all ages being 128·91, and Goepp with 9,996, his average for all ages being 123·1, but both take slightly different age groups, which prevent inclusion in the above table. In Fisher's report only 13 per cent. of entrants are younger than thirty-six, so that his general average is high. In Mackenzie's and Goepp's tables the largest number of entrants was about age thirty, so that their general averages are low. Goepp's examinations were made in 1918 under war conditions, particularly in the matter of eating and the

TABLE V

Average Differential Pressures—Men only (Symonds)

Ages.	Average Totals.
15—19 . . .	42·9
20—24 . . .	43·0
25—29 . . .	43·0
30—34 . . .	41·8
35—39 . . .	42·2
40—44 . . .	42·3
45—49 . . .	43·1
50—54 . . .	45·2
55—59 . . .	46·7
60 and over . .	49·2
6,071 Entrants.	42·8 All ages.

use of alcohol, which may have lowered the general and individual age period averages.

By reviewing these tabulated figures with large numbers of my own records I have drawn up the following theoretical table corrected to represent standard arterial pressures at various ages for men of medium physique (Table VI.).

TABLE VI

*Theoretical Standard Arterial Pressures in Males of Medium Physique at various Ages**

Age in Years.	Systolic Pressure.	Diastolic Pressure.	Differential Pressure
At birth	20—60 (average 40)	?	?
Up to 2	80	48	32
5	88	55	33
10	100	64	36
15	110	70	40
20	122	80	42
25	124	81	43
30	126	82	44
35	127	83	44
40	128	84	44
45	129	85	44
50	130	86	44
55	132	87	45
60	135	89	46
65	140	90	50
70	143	92	51
75	147	93	54
80	150	94	56
Over 80	All pressures tend to fall.		

* Pressures estimated by auscultatory method with Riva-Rocci or Tycos types of instrument. In children under the age of seven years the diastolic point is difficult to determine.

Athletes often give readings below standard, owing to the effect of training in promoting a vigorous peripheral circulation. At times the systolic pressure may be as low as 100 to 105 mm. As a result of greater vasodilatation, low pressures are alleged to be the rule in tropical countries.[77]

Tables, though handy for reference, are hard to memorise. Hence, whenever one comes across a case in which the blood pressure is out of the ordinary, it is desirable to be able to apply some easy rule for finding out what is approximately the standard pressure for a given age. The popular idea that the systolic pressure of any adult can be arrived at by adding 100 to the age in years is erroneous and most inaccurate, particularly for all ages over forty years. It is negatived by *post-mortem* evidence as to the weight of the heart in hyperpiesis as compared with records of the arterial pressure during life, for, in both sexes, greatest increase in heart weight does not coincide with the greatest age (p. 105).

A closer approximation is afforded by taking standard systolic pressure as 100 plus half the age, and standard diastolic pressure as 75 plus one-quarter of the age.

It must, nevertheless, be realised that no mechanical formula is likely to be as exact as the above theoretical table. The two rules which the author finds best and most serviceable in practice are here appended :—

1. **Simple Rule for calculating Standard * Diastolic Arterial Pressure** (Halls Dally).—*Taking standard diastolic pressure at age twenty as* 80 *mm. Hg, for each five years above twenty up to and including age sixty add* 1 *mm. Hg, and for each five years above sixty up to and including age eighty add* 2 *mm. Hg.*

2. **Simple Rule for calculating Standard * Systolic Arterial Pressure** (Halls Dally).—*For ages twenty up to sixty, standard systolic pressure equals* 120 *plus one-fifth of the age. At age sixty standard systolic pressure is* 135, *and for each year above this up to and including age eighty add* 1 *mm. Hg.*

Standard differential pressure between ages fifteen and twenty-five is 43 mm. Hg ; between ages twenty-five and fifty, 44 mm. Hg.

* Standard = " normal," *i.e.*, non-pathological.

Limits of Normal Arterial Pressure Variation

Systolic arterial pressure may vary 15 mm., and diastolic pressure 10 mm., *above or below* the figures given in Table VI. for various ages without such deviations being regarded as necessarily abnormal.

The lowest normal limit of systolic pressure may be regarded as—in young children, 70 mm Hg; in adolescents, 85 mm. Hg; in adults, 105 mm. Hg.

For working purposes it may be said that "normal" arterial pressures for young and healthy adults are as follow: the systolic pressure may vary between 105 and 125, the diastolic level is usually between 65 and 80 mm., and the differential pressure 42 to 44 mm. The figures usually met with under normal conditions from middle age onwards range from 130 to 145 mm. Hg.

Influence of various Physiological Factors on Arterial Pressure

Body Weight, Age, Height and Sex.—Arterial pressure varies directly with weight and height, as a rule increasing with increasing weight, and *vice versâ*. Height has a similar, but less, influence. Larimore[78] found that in 417 factory hands the sthenic habitus was accompanied by a higher arterial pressure than the asthenic The pressure in the hyposthenic habitus was intermediate.

Examination of 200,000 males, whose lives were insured between 1887 and 1908, and whose histories were followed till 1921 or prior to termination of the policy, has shown that danger to life increases directly with the degree of overweight. The relation between age and weight is also noteworthy. After age forty-five excess mortality among overweights becomes of great importance. Those who are more than 25 per cent. overweight have a mortality rate double the normal (L. I. Dublin). Nevertheless, the normal rise in arterial pressure during the second half of life, as evidenced by mass statistics, does not amount to more than 15 mm. Hg.

In women up to the age of forty years, standard diastolic and systolic pressures are a few millimetres lower than the

above figures for men. After this age in women, standard systolic pressure becomes equal or higher, while diastolic pressure for the next ten years remains about the same. At the age of fifty, standard diastolic pressure increases rapidly, and afterwards is substantially higher than that for men. Perhaps menstruation is responsible for the lower range of pressures in women below forty, but it does not seem reasonable to assign the menopause as the cause of higher pressures after fifty. Note that systolic and diastolic pressures in both men and women show a definite increase with weight as well as with age.

Sleep.—During quiet sleep there is a fall of from 10 to 20 mm. or even more in the systolic pressure, with a fall also in the diastolic, both as a result of lessened irritability of the nervous system, muscular relaxation, and vascular dilatation, the pulse rate being slowed by twenty beats per minute. The pressures are lowest during the first few hours, and gradually rise again towards the hour of waking. During disturbed sleep the systolic pressure may rise from 50 to 70 mm., and the diastolic by 30 mm. Hg, which accounts for the incidence of cerebral hæmorrhage during sleep in about 20 per cent. of hyperpietics.[79]

Rest.—The combination of muscular relaxation and mental quietude produces similar effects to those observed during sleep, though to a less degree.

Posture.—Mortensen [80] found on examination of several hundred adults of both sexes that passive postural change from the reclining to the standard position caused slight decrease in systolic pressure and slightly greater rise in diastolic pressure, thus cutting the differential pressure at both extremes. There was also a uniform increase in pulse rate. In very few cases was the systolic pressure higher in the erect posture, and in no case was the diastolic lower. The findings of Max Ellis [81] in the main correspond with these results.

Food.—The influence of food intake on blood pressure is not yet definitely established. Probably immediately after meals a slight rise which results from gastric filling occurs in systolic pressure, accompanied by a constant fall in diastolic pressure, even up to 20 mm., independently of the time of

day. This is speedily succeeded by a drop in systolic pressure to below standard level, coincident with the period of active stomach secretion. Finally, there is another rise probably due to intestinal distension with food.

Fasting.—Fasting induces a gradual fall in both pressures which rapidly return to normal when the fast is broken.

Alcohol.—(1) Experiments show that, if alcohol raises blood pressure at all, such rise is transient, depending on temporary reflex vasoconstriction in the splanchnic area through the afferent fibres of the gastro-intestinal surface, and that, as soon as alcohol reaches the blood stream, peripheral vasodilatation results, and the pressures fall. Intravenous injection of alcohol causes a drop in blood pressures without preliminary rise.

(2) Donnison's [82] observations on African natives support the view that hyperpiesis and arteriosclerosis are evils connected with civilisation and mental stress, since in 1,800 natives investigated by him no clinical case of raised arterial pressure nor of arteriosclerosis was found. Yet, to my knowledge, there are few races, however primitive, that do not brew some form of alcoholic beverage.

(3) Hyperpiesis and arteriosclerosis are by no means necessarily coincident, *i.e.*, in only about 50 per cent. of cases of established arteriosclerosis are arterial pressures above normal.

(4) Of 16,662 policy holders of the New York Metropolitan Life Office, 19·8 per cent. manifested abnormal arterial pressures, approximately 16 per cent. being high. But the percentage of high pressures in excess alcoholics was little if any higher than for the others.

(5) In habitual drunkards the incidence of arteriosclerosis has repeatedly been found to be not more than normal, and Ruffer,[83] who performed over 800 autopsies on Mohammedan pilgrims who had never imbibed alcohol, found in them arterial disease as frequent and as early as in individuals who practise no such abstinence.

(6) The actual amount of alcoholic consumption per diem must always be taken into consideration when assessing the possible effects. The issue is often confused by those who class together light wines and beer, which contain very

little alcohol, with heavy beers, wines, spirits and liqueurs, whose alcoholic content is far heavier.

Hence there is no direct evidence to connect alcohol either with the production of hyperpiesia or arteriosclerosis.

In this connection a tale told by the late Sir James Mackenzie is illustrative. An elderly brewer's agent consulted him because of symptoms of slight angina pectoris. Hyperpiesis of 210 mm. systolic, with a very hard pulse and thickened arteries, were found. Sir James said to the patient, "You must give up beer and spirits." The reply was, "I'm a teetotaller." "Well then, you must eat less butcher's meat." "I'm a vegetarian," was the answer!

Tobacco.—Like alcohol, the immediate effect of smoking is a pressor one, which raises both pressures, the maximal more than the minimal, and quickens the pulse rate by about twelve beats per minute. In respect of both alcohol and tobacco it should, however, be remembered that experimental dosage is much greater than the amounts habitually absorbed by man, and that in drinks the percentage of alcohol varies enormously.

Tobacco smoke contains pyridine bases and nicotine, the latter being the main toxic product. The composition of tobacco smoke varies greatly both with the kind of tobacco and the manner of smoking. Most of the nicotine is usually burnt up, and that which reaches the mouth is volatilised by the hot gases in passing over the unburnt area. A thick cigar has the worst effect, since it acts as a chimney for the gases which in a thin cigar or cigarette escape into the surrounding air. In long-stemmed pipes much of the nicotine condenses before reaching the mouth. Inhaling the smoke, however, more than compensates for this difference in combustion.[81]

In non-smokers the immediate effect of smoking is to send up systolic pressure by 10 to 30 mm., the diastolic being much less influenced. Within five minutes or so systolic pressure drops often by 50 mm., with palpitation, pallor, sweating, colicky abdominal pains, *muscæ volitantes* and weak pulse.

In those more habituated to the use of tobacco subacute symptoms occur only from over-indulgence, or from smoking

a kind of tobacco to which they are unaccustomed. In seasoned smokers little or no rise in systolic pressure results.

Generally speaking, smoking at first increases systolic, diastolic and differential pressures, though later these return to normal Excessive smoking lowers the systolic pressure. Middle-aged smokers are liable to precordial stabbing pains and angina pectoris, which disappear on giving up the habit. In the elderly, secondary arteriosclerosis and myocardial changes may be induced.[18]

Cornwall[85] has made a series of observations upon the effect of chronic tobacco poisoning on blood pressure. Patients with symptoms of tobacco heart had subnormal blood pressures, the systolic ranging between 85 and 115 mm. Hg, the diastolic between 60 and 75 mm. Hg; the pulse rate was not necessarily rapid, notwithstanding the presence of cardiac symptoms. The systolic pressure was generally lowest when the subjective symptoms were most pronounced, whilst brisk exercise caused a uniform fall in both systolic and diastolic pressures. A systolic pressure of less than 100 mm Hg in an otherwise healthy man is almost certainly due to excessive smoking. If there is no evidence of this, careful search should be made for symptoms and physical signs of active fibro-caseous pulmonary tuberculosis.[86] As age advances, and as arteriosclerosis becomes more frequent, the tolerance for tobacco becomes less.[87] Cigar-smokers are more prone to arteriosclerosis than cigarette or pipe-smokers. Excessive cigarette smoking causes cardiac irritability, but for most people *moderate* use of tobacco does not appear to be injurious.

Exercise.—In healthy subjects, increase in blood pressure accompanying physical exertion is in direct proportion to voluntary effort required, and to amount of work done. The rise in pressure mainly results from (*a*) increased cardiac energy, (*b*) mental concentration. Unaccustomed kinds of exertion which involve mental effort produce a much greater rise than is caused by an equivalent amount of routine work, as, for example, the flushed face of one learning to cycle, or of a young barrister conducting his first important case. Both psychical and

physical factors influence systolic pressure more than diastolic.

Mild muscular exercise, such as walking, tends to lower the minimal pressure whilst augmenting the blood flow, pulse rate and differential pressure. Foot racing in healthy athletes causes a considerable reduction in both systolic and diastolic pressures, the differential remaining almost contant. More strenuous exertion increases all of these, and, if very severe, the initial rise of pressure may be extreme, particularly in those unused to manual labour, through lack of automatic adaptation to such efforts. Diastolic pressure may be raised by even as much as 30 mm. temporarily, and systolic pressure by as much as 70 mm. In men given to arduous muscular work, arterial pressures, tested during the intervals of rest, range habitually below the average.[61]

Altitude.—The effect of altitudes of 4,000 feet or above on the pulse rate of normal individuals is to cause an initial and temporary acceleration lasting for about ten days, after which the pulse rate gradually falls. Those acclimatised to high altitudes manifest pulse rates which are slow, readings of from 54 to 60 beats per minute being commonly met with.

Fatigue.—If exertion be so prolonged and excessive as to trench upon the reserve power of the heart, fatigue ensues. After a transitory rise of pressure, mainly systolic, due to an effort on the part of the vasoconstrictor centre to maintain the pressure head, the centre becomes depressed, with resultant fall of both maximal and minimal pressures. The pulse rate diminishes, and dyspnœa and other subjective symptoms appear.

Psychical Stimuli.—Fear, anger, emotion, worry, anxiety, pain and mental activity exert through vasoconstriction a pressor effect, and bring about sudden changes in systolic pressure. Under stress of emotional crises there may be an extraordinary and rapid rise in association with notable quickening of the heart rate. Nervous individuals are more susceptible than those of placid temperament. In normal persons it is usually only the systolic pressure that varies, whilst in the arteriosclerotic the diastolic fluctuates to a similar extent.

The following history, told me by a general practitioner,

is of psychological interest, since it illustrates the dramatic rise which can occur in systolic pressure as the result of sudden and strong emotion, as well as emphasises the importance of not being content with one observation of blood pressure only :—

The patient was a man aged fifty-seven, whose shipping business necessitated his working at high pressure and making frequent long railway journeys. On arrival at Paddington one evening, feeling tired out, he went to the refreshment room and had two glasses of brandy-and-soda. He then took a train to his home, some eighteen miles from London, and on arrival went straight to bed and sent for the doctor.

The doctor found his patient restless and excited, with flushed face and complaining of throbbing headache, the pulse of 90 per minute being full and bounding. The average of three blood pressure readings was 265 mm. systolic. The urine was found to be normal. A strong dose of calomel was given, and cooling applications made to the head.

On the following morning, to the doctor's surprise, the patient appeared calm, cheerful, and free from all symptoms.

The patient, noting the doctor's astonishment, asked the reason, and was told of the rapid fall in blood pressure since the night before. Having again found the urine normal, the doctor suggested that a "sudden shock" might have caused the condition, whereupon the patient told him that he had married late in life a wife thirty years younger than himself. Previous to marriage he had been a hard liver and heavy drinker, but after a long and terrible fight became a total abstainer. From that day no alcohol passed his lips until the evening before. On letting himself into the house, feeling still tired out, he was met by his wife, who asked: "What's the matter?" and detecting the odour of spirit, exclaimed: "At your old drinking habits again, are you?"

The memory of the agony through which he had won and the realisation, which then became acute, that he had made a mistake in marrying a wife so much younger than himself, together with the tone in which she spoke, just seemed the breaking point. His head felt as if it would burst; he clung to the side of the stairs to prevent himself falling, and sent for his doctor.

Normal Puberty, Menstruation, Pregnancy, Labour and the Menopause.—(*a*) *Puberty.*—From very early ages there is a progressive steady rise of arterial pressure up to the onset of puberty, then a quickening of the rise up to the adult level somewhere between the ages of seventeen to twenty years.

(*b*) *Menstruation.*—During the seven to nine days preceding the menstrual flow both pressures rise. With the onset of the period they rapidly fall to reach their lowest

levels at its end. During the next fortnight pressures gradually return to their previous standard levels.

(*c*) *Normal Pregnancy.*—In primiparæ below thirty years of age as a rule one does not expect to find systolic pressures of more than 95 to 115 mm. Hg, with diastolic pressures respectively of from 50 to 75. In multiparæ, probably as a result of increased age, pressures range somewhat higher, from 115 to 125 mm. systolic, and 60 to 80 diastolic respectively. About 60 per cent. of all cases fall within a maximum of 125 systolic. Thus, as a result of general experience, one may say that, during the whole term of normal pregnancy, arterial pressures are within standard limits, having regard to the fact that the range of arterial pressures in women is lower than that for men of corresponding age. Initial lower ranges of pressure gradually give place to higher ones. During the last three months, however, there is usually a slight and gradual rise of arterial pressure in conjunction with a definite increase in basal metabolic rate. This latter is in all probability not wholly due to growth of the fœtus, but also to enhanced thyroid activity.

(*d*) *Normal Labour.*—Arterial pressures should be estimated before, during and after delivery. Any rise at the beginning of and during normal labour is brought about solely by pain, or by voluntary and involuntary muscular exertions due to labour. On subsequent relaxation, pressures gradually drop during convalescence to normal levels.

(*e*) *Normal Menopause.*—The normal menopause should be unassociated with arterial pressure variations, but in the majority of all cases there is a rise in the maximal and differential pressures (*vide* p. 180).

CHAPTER VI

Hyperpiesis : Hyperpiesia : Hypertonia

"In primary contracted kidney hyperpiesis is but an incidental coefficient, while in hyperpiesia on the contrary it would seem that it is the high systolic and diastolic pressures themselves that do the mischief, or most of it"

Allbutt: *Arteriosclerosis*, 1923.

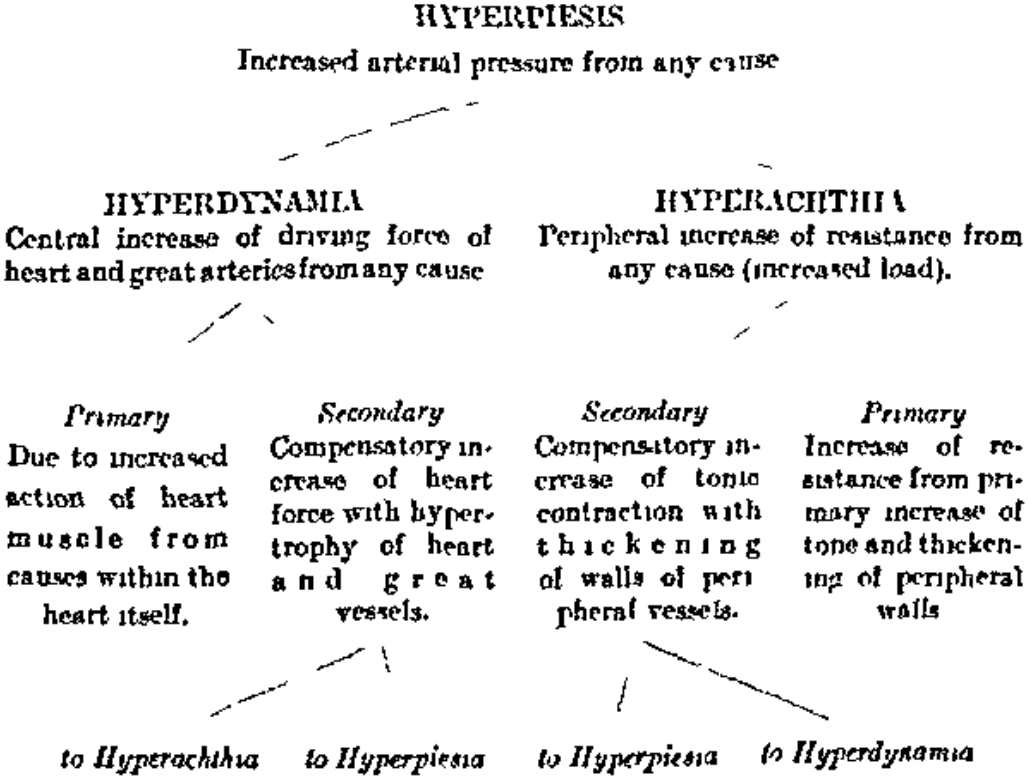
I. *Hyperpiesis*

Hyperpiesis (Gr. *ὑπέρ, πίεσις* = over-pressure), in its application to the arterial system, means high arterial pressure, *i.e.*, a rise of pressure within the arteries from any cause whatever, whether of renal or non-renal origin, temporary or permanent, physiological or pathological.

Starting with this definition, we are thus enabled to translate the physical principles of the circulation (p. 71) into their equivalents and so formulate a classification according to Table VII.

Table VII

Classification of Hyperpiesis



In physics all pressures are expressed in terms of force and load, so that, in order to avoid the repetition of long and cumbersome phrases, I have introduced as corresponding physical expressions two new words, hyperdynamia (ὑπέρ, δύναμις = over-action) and hyperachthia (ὑπέρ, ἄχθος = over-load). Hyperpiesia is maintained in its original signification.

On this classification hyperpiesis constitutes the chief heading. This includes two subsidiary divisions, hyperdynamia and hyperachthia.

Primary hyperdynamia results from any cause which necessitates the driving power of the heart and great arteries *being augmented, and in time leads to hypertrophy.*

Secondary hyperdynamia results from hyperachthia, *i e.*, increased load, which causes the heart and great vessels to hypertrophy, *e.g.*, a damaged kidney produces secondary hyperdynamia in the rest of the circulation. Similarly arteriosclerosis causes secondary increase in the force of the pump because of the greater difficulty met with in driving the blood through sclerosed arteries—again a secondary hyperdynamia

Primary hyperachthia, or increase of load, results from any cause which increases peripheral resistance, and leads to hypertonia, *i.e*, an increased contractility of vessel walls, which in turn, if long continued, induces vascular hyperplasia.

Secondary hyperachthia occurs in response to a primary or secondary hyperdynamia, as in the case of a primary arteriosclerosis.

Secondary hyperdynamia and hyperachthia respectively result from hyperpiesia acting either centrally or peripherally. Even over long periods of time it is surprising to find that little change may have occurred in the heart, this organ being affected solely by increased load. In the majority of established cases of true hyperpiesia, nevertheless, the heart becomes overworked and hypertrophies to meet the pressure, as evidenced clinically by some degree of enlargement, which can be confirmed by X-ray investigation, by more rapid action (sometimes amounting to tachycardia) and by lessened response to effort. Still later, the

vessels of the periphery become thickened for a similar reason in a combined process of degeneration and repair.

Thus the circle is completed, as shown in Table VII., which includes all varieties of hyperpiesis, and can readily be adapted for low pressure states by the substitution of "hypo" for "hyper."

Range of Pressures in Hyperpiesis.—Any diastolic pressure of 105 mm. or over, and any systolic pressure of 160 mm. or over, may be regarded as hyperpiesis, which, as stated previously in other words, is not *essentially* a pathological condition.

General Frequency.—Of the total deaths from all causes in England and Wales, diseases of the cardio-vascular system account for more than one-third. In this group approximately one-fifth show evidences of high arterial pressure.

Age Incidence.—Hyperpiesis may become manifest at any age, most frequently between the ages of fifty-five and seventy years. Below the age of forty it is relatively uncommon.

Sex Incidence.—Women are affected in slightly less degree than men except at the climacteric

Operative Risks.—As a rule hyperpietic patients stand operation well. The opposite is often the case with subjects of low arterial pressure.

Variability.—One striking fact concerning hyperpiesis is its extreme variability. Apart from the action of drugs, within a few days it is capable of dropping from a great height to a point only slightly above standard. The opposite phenomenon equally may obtain. The association of arteriosclerosis, which is a constant and stable condition, cannot be invoked to explain these interesting fluctuations, but a variable amount of circulating toxin might readily account for changes in the media as well as the small-cell proliferation in the intima.

Characters.—In well-established hyperpiesis, "whether renal or not, the vessels are tightened with blood dammed back. The heart and main vessels are so full that the output of the ventricle against the resistance is less and less, while distension of the arteries is pushed towards the outward limit of their elasticity; thus their further function, as an

elastic reservoir between heart and capillaries, of forwarding the blood during cardiac diastole is checked, and diastolic pressure rises." [88]

The Heart-weight in Hyperpiesis.—From an analysis of 7,000 *post-mortem* cases occurring in twenty consecutive years at St. Bartholomew's Hospital, and from a further series of thirty-two cases,[89] Dr Geoffrey Evans finds that a heart-weight of 15 oz. or over has some definite relation to chronic interstitial nephritis or cerebral hæmorrhage, whereas a heart-weight of from 12 to 14 oz. may occur in a great variety of other non-related conditions. "Idiopathic" cardiac hypertrophy, *i.e.*, not due to disease of heart and lung, of 15 oz. *post-mortem* is thus a direct indication of diffuse hyperplastic sclerosis, evidenced clinically by a systolic pressure of 180 mm or more. In this group within the limits imposed by certain factors, *e.g.*, size of body, physique and adiposity, the measure of the degree of hypertonia may be stated as an increase of 1 oz. in heart-weight for each 10 mm. rise in arterial pressure.

For the reason that the walls of the remaining three chambers of the heart may hypertrophy, especially if, as a result of leakage of the mitral valve, slowing of blood flow occurs in the pulmonary circuit, Batty Shaw [90] is of opinion that left-sided ventricular hypertrophy is a better gauge of the direct effect of hyperpiesis upon the heart than the total weight of the organ. He gives a series of cases, twenty-nine men and six women, with hyperpietic hypertrophy of the left ventricle uncomplicated by valvular endocarditis, the average weight of the whole heart for the men being 18 oz. (normal 11 oz.), and for the women 14 oz. (normal 9 oz.). Greatest increase of cardiac weight was not found at the greatest ages in either sex. This is explained by the weakening of all muscles, together with lessening of tissue resistance, which coincide with advancing age.

II. Hyperpiesia

Hyperpiesia.—From *hyperpiesis*, which includes high arterial pressure arising from any cause, it is useful to separate out *hyperpiesia*, a condition synonymous with the

so-called "essential arterial hypertension," a cumbrous and hybrid term now unfortunately employed at home as well as abroad (p. 78).

Nature of Hyperpiesia.—As described by Allbutt, hyperpiesia is a clinical morbid series characterised by raised arterial pressure in association with hypertrophy of the heart and changes in the vessels, distinct from the recognised forms of Bright's disease.

It has been suggested that one of the primary causes of hyperpiesia may be found in a heightened viscosity of the blood, resulting either from a primary albuminæmia—a colloid phenomenon—or from mixed colloid and crystalloid phenomena differing in degree in individual cases Heightened blood viscosity is the rule in polycythæmia, but of 66 out of 189 collected cases of this malady investigated by Lucas,[91] in which records of arterial pressure had been preserved, in 21 (approximately one-third) the systolic pressure was under 140 mm. Hence hyperpiesia appears neither to be caused by polycythæmia nor by increased viscosity of the blood.

Associations with other Conditions

Other accompaniments of hyperpiesia include proteinuria and ocular changes. There are also associations with such widely differing conditions as exophthalmic goitre, asthma, gout, lead poisoning, infections of and hæmorrhages into various organs and tissues, paralysis, and other conditions too numerous to mention.

Batty Shaw's[90] exhaustive study of forty-seven cases of hyperpiesia compels him to believe that hyperpiesia and eclampsia are closely allied toxic states, that both may be associated with renal disease and retinal changes, and that both may be temporary and recovered from, or may be permanent and fatal The early symptomatology suggests that the primary causes of hyperpiesia are either psychical or toxic.[92] [93] [94] [95] Endocrine imbalance and vasomotor instability are thus probably secondary, an over-responsive endocrine-autonomic system with attendant vasomotor instability in the direction of vaso-constriction being due in

many cases to overaction of the posterior pituitary as a result of long-continued psychical stress or sudden and violent emotional strain.

Clinical Features.—Hyperpiesia constitutes *a state of primary high arterial pressure* in association with a distinct train of clinical and pathological events, recurring in many persons with such frequency of association and consistency of course as to constitute a disease, which, arising independently of arteriocapillary, renal, cerebral or other demonstrable morbid change, after a variable period of latency, to which no definite term can be assigned, clinically manifests individual characters, the chief of which are (1) high diastolic and systolic pressures, persistent but not necessarily permanent, (2) left ventricular hypertrophy. *On X-ray investigation the heart is found to be enlarged*, and an electrocardiogram shows left ventricular preponderance.

Hyperpiesia is among the commonest of diseases. It is wont to occur in subjects above middle age in both sexes, more often in men, and mostly between the years from fifty to seventy. The onset is insidious, for in most cases no date can be assigned to the initial changes, which may have begun at least twice as long ago as the time at which the condition was first recognised For variable periods the subjects may have fair or even robust health, slight symptoms either having been ignored, or more usually ascribed to causes other than the actual, so that by the time that the patient seeks medical advice, the condition is already well established.

Types of Hyperpiesia.—Two forms are recognisable—labile and stable. The early labile form [96] presents a curve of wide daily fluctuations in pressure, though the general average level is definitely high. The lowest levels, often to normal values, *occurs only during sleep.* For a true picture of this condition, an instrument recording continuous pressures is necessary. This notable instability is more characteristic of the labile form of hyperpiesia than the actual heights of pressure recorded. The later stable form, as the name implies, shows high levels with much slighter variations, and few symptoms, whereas the labile form has many symptoms.

The majority of cases have an abnormally low sugar tolerance, which may depend upon psychical disturbance leading to a lack of endocrine balance, glycosuria, which is at times associated, depending upon this and not upon disturbance of pancreatic function.

In pure hyperpiesia, moreover, there is no evidence of blood stasis in the capillary circulation.

Among certain authors there has been a tendency to divide hyperpiesia into various types, such as paroxysmal, benign and malignant. The paroxysmal type is characterised by temporary pressure elevations above a constant high level, the "malignant" is probably a more rapid and severe form of the same pathological picture that occurs in the "benign," usually terminating by uræmia as a result of renal failure.

Irritative influences, which upset circulatory balance, whether of toxæmic origin or otherwise, tend to induce a state of ill-health, increasing the cardiac load and thus leading to cardiac failure, while the production of arterial thickening and subsequent degeneration disposes to apoplexy, renal involvement and other structural changes.

Pathology.—A. The Vasomotor Centre in the Medulla.—Most observers are in agreement that the blood pressure raising stimulus, however induced, has to do with the controlling vasomotor centre in the medulla oblongata. Such stimulus has been attributed to a heightened sensitiveness,[97] or alternatively, to arteriolar proliferation, claudication or toxic damage of its capillaries, or interference with oxidation,[97] or to arteriolosclerosis of the medulla.[98] Arguments have been advanced, however, against both the latter suppositions.[99] [100, 101]

B. The Autonomic-Endocrine System.—Under the direction and control of the medullary vasomotor centre, regulation of the circulation is largely vested in the autonomic endocrine system. The autonomic (vegetative) nervous system consists of two physiologically antagonistic divisions, namely, the sympathetic system (katabolic) and the parasympathetic or extended vagus system (anabolic).

The sympathetic acts with the pituitary, thyroid, adrenals and gonads, exerting also a considerable influence upon

metabolism in general, while the parasympathetic has more limited associations with the parathyroids, and with the cell-islets of the pancreas over which direct vagus control has been proved.

In turn the endocrine glands activate or depress the circulatory mechanisms *viâ* the sympathetic nervous system by means of their chemical messengers (hormones), a pressor influence being exerted by vasopressin (posterior lobe of pituitary) and by adrenaline, while the cell-islets of the pancreas furnish the depressor hormone, vagotonine.

C. Influence of the Pituitary Gland.—For many years the thyroid has been regarded as the master gland and activator of the endocrine series. Recent experiments on amphibia and mammals, however, go to prove that the differentiation and continued functioning of the thyroid depends on a thyroidotropic hormone of the anterior lobe of the pituitary, and the functions of the pituitary so far discovered suggest that it should now be held to supersede the thyroid as the dominant member of the ductless chain in bringing to structural and physiological fruition the thyroid, parathyroid suprarenals and gonads, thus exerting general effects upon the body, and through the thyroid influencing metabolic rate.

This new and striking conception is strongly supported by the recent work of Harvey Cushing.[102] From this it appears that the central representation of both sympathetic and parasympathetic systems lies in the hypothalamus, between which and the neighbouring posterior lobe of the pituitary (neurohypophysis) there is a definite nervous connection. Cushing states that the posterior pituitary lobe possesses an active principle or secretion capable, among other properties, of raising arterial pressure and of diminishing renal secretion. This active principle is derived from the pars intermedia, whose cells, when ripened, become basophilic, invade the pars nervosa and become transformed into hyaline bodies which make their way towards the infundibular cavity and extrude themselves into the third ventricles, in the fluid of which this secretion of the posterior lobe may eventually be detectable. In other words, the basophile cells normally present in the anterior lobe and

pars intermedia of the pituitary migrate into the posterior lobe and thence to the third ventricle.

In hyperpiesia, and in certain toxæmic states, *e.g.*, eclampsia, characterised by raised arterial pressure, a substance indistinguishable in its effects from posterior lobe extract has been found in the blood stream, but not detectable therein under normal conditions (Anselmino [103]). The same or a similar substance has been found in the blood of certain patients with hyperpiesia (Irvine Page).

From the above, Cushing derives the conclusion that infiltrative basophilia of the pars nervosa of the pituitary gland is an expression of functional overactivation of the posterior lobe, and represents the pathological basis of these hyperpietic disorders.

D. Influence of the Adrenal Glands.—The next step is to determine as far as possible how this stimulus to pituitary overaction is brought about.

Excessive secretion of adrenaline has been invoked by Vaquez,[104] Josué [105] and Paul [106] as a cause of raised arterial pressure. In support of this belief various reasons may be adduced :—

(*a*) " Preganglionic sympathetic fibres end in the adrenal gland, whose medullary cells *are* the sympathetic ganglion cells, and we have here an interesting example of a nervous structure assuming secretory properties. It is of striking interest to find, as Langley showed, that their secretion, adrenaline, has the same effect on any part as stimulation of the sympathetic nerves to that part." [107]

Thus the adrenal glands are closely related to the sympathetic, and the effect of adrenaline on any part is to heighten the sensitiveness of response to the sympathetic in the same way as by stimulating its sympathetic nerve, which in turn increases the flow of adrenaline into the circulation. Reaction to infection and to elementary emotional experiences has been explained as an emergency adrenaline response of protective character. The reciprocal associations of the thyroid with the sympathetic are similar, but katabolic. Many of the high pressures which I saw in soldiers invalided for " disordered action of the heart " were of this type, sustained endocrine-sympathetic reaction to

the fear sense being evidenced by the staring eyes, sweating, quick pulse and tremors, resembling the acute stage of *hyperthyroidism.*

(*b*) Healthy human adrenal glands together contain approximately 8 mg. adrenaline, an amount which in high pressure states is increased in conjunction with hyperplasia of the adrenal medulla.[108] Bru[109] injected 10 c.c. anti-medullary serum intravenously into dogs, and found within five minutes a rise in arterial pressure which lasted for over ten minutes.

(*c*) Although in hyperpiesia the presence of adrenaline in the blood has not been established, it is, nevertheless, probable that a flow of adrenaline into the blood from the cells of chromaffin sympathetic tissue in the adrenals and elsewhere exerts an influence upon the adjacent sympathetic system. An analogy exists in the autonomic system in which an "autonomin," vagotonine, has been isolated by Santenoise[110] in pure form from the internal secretory portion of the pancreas.

(*d*) *Primary tumours of the adrenal cortex have been* found occasionally attended by a permanent hyperpiesis, and the rare adrenaline-secreting adrenal medullary tumours give rise to paroxysmal or permanent hyperpiesis. The subjects of chromaffin tumours have manifested paroxysmal hyperpietic crises.

Discrepancies noted by numerous observers between the extent of pathological changes in the adrenal glands and the clinical appearance of hyperadrenalæmia, giving rise to so much controversy and for which no satisfactory explanation has been forthcoming, may, however, be rationalised if it be remembered that *secretion of adrenaline is not limited merely to the adrenal tissue, but is shared by other islets of chromaffin sympathetic tissue scattered throughout the body.*[111]

On the other hand, Cushing[102] has adduced more recent evidence that certain reported cases of cortico-adrenal hyperpiesis are examples of basophile adenoma of the anterior lobe of the pituitary gland. Out of eighty patients with various forms of hyperpiesis treated by Abrami, Santenoise and Bernal[112] with vagotonine, usually by

hypodermic injection, thirty derived permanent benefit, the best results being obtained in those with paroxysmal crises of hyperpiesis, while several patients did not react. "These results, taken in conjunction with the paroxysmal crises of high blood pressure in patients with chromaffin-celled tumours of the adrenals, lend support to the view that essential hypertension is not due to excess of adrenaline in the blood." [113]

E. Other Pressor Substances.—Considerable endeavours have been made to isolate other pressor substances in the blood, due to altered metabolism or to inadequate excretion, particularly of protein derivatives (*vide* Chapter VIII).

Since 1924 Major and Stephenson [114] have studied the probable *rôle* of guanidine compounds in the production of raised arterial pressures, and have determined the daily output of guanidine bases during the period of falling arterial pressure. This fall coincided with a gradual rise in output of dimethyl guanidine, the rise being maintained for several days after the arterial pressure decline, thus suggesting that increased diuresis eliminates an excess of pressor substance which has been retained in the body.

In about half the number of hyperpietics in a large series investigated, Major [115] found an increase in the blood of a substance with reactions similar to those of guanidine. These results have received confirmation from Pfiffner and Myers.[116]

Other non-protein nitrogenous substances such as creatine and creatinine appear to have no effect in raising arterial pressures.

Since tyramine may be produced *in vitro* from tyrosine by the action of fæcal bacteria, it is not unreasonable to suppose that the presence of this substance in the large bowel and its absorption therefrom may promote pathological conditions of which heightened arterial pressures constitute a prominent symptom. As in the case of adrenaline, lengthy administration of tyramine induces renal and vascular lesions akin to those which so generally accompany persistent hyperpiesis in man. Further, it is of interest to note that there is a relationship between the amino-acid group, the blood nitrogen urea, and the diastolic pressure so that, if

one can control the diastolic pressure by checking absorption of pressor amines, systolic variations are of less moment.

TABLE VIII

Differential Diagnosis between Hyperpiesia and Chronic Glomerular Nephritis

	Hyperpiesia	Chronic Glomerular Nephritis
History	No definite antecedents	Preceded by subacute nephritis or long latent stage.
Appearance	Robust, often plethoric	Variable, sometimes pallid
Age	50–70 years Rare under 40	Usually under 40 years
Arterial pressure	May be very high, 250 + Labile, often falls with rest	Moderate or high. Stable.
Retinitis	Uncommon	Frequent.
Renal function tests	Normal, or slight changes	Reveal inadequacy
Urinary findings:		
Volume	Augmented.	Augmented, diminishing at the last
Specific gravity	Low: may be fixed	Low and fixed.
Albumin	Absent or slight trace	Slight in early stage, abundant in late.
Casts	Absent usually.	Scanty, hyaline or fatty
Red blood cells	Absent	Absent without acute exacerbation
Nyctuna	Absent	Present
Blood findings:		
Nitrogen retention	Absent.	Present
Anæmia	Absent or slight	Present
Uræmia	In about 8 per cent. of cases	Frequent

III. Hypertonia

Hypertonia (*Angiospasm*) *represents an active state of the* smooth muscle in the walls of arteries and veins, in contradistinction to high arterial pressure (hyperpiesis) which indicates the lateral pressure exerted by the blood upon the vessel wall.[117]

Normal arteries are barely palpable, but in hypertonia both qualitative and quantitative changes in hardness occur. Arterial hypertonicity, in larger or smaller areas, is met with in a host of disorders. *In some the hardness* persists; in others it may lessen with time. To realise that

long-hardened arteries may suddenly become soft, one has only to study the vessels of the paralysed side in hemiplegia. Since hardening may coexist with weak cardiac action, there can be no necessary connection between hardening and increase of blood pressure, though, in general, increased tonus is linked with increased pressure, and is compensatory.

Owing to the presence of smooth muscle in the walls of the veins, hypertonia, and even hypertrophy, may also occur in these.

Arteries may vary considerably in calibre (*vide* "Tonus," p. 77). Narrow rather than wide is the rule in hypertonic vessels. A narrow hypertonic artery feels very different from a wide one, yet the former is found in secondary contracted kidney cases, while the latter may at times be met with in association with arteriosclerotic kidneys.

To some extent raised arterial pressure may be differentiated from arterial hypertonus by instrumental methods, yet the varying degrees of each cannot be measured independently (p. 115, B, *b*).

Associations of Hyperpiesis, Hyperpiesia and Hypertonia (Vascular Hypertonus), with Arteriosclerosis and Renal States.

Persistently raised blood pressures do not *necessarily* connote anatomically altered arteries, though presumably the muscle elements manifest some increase in number and size. That hypertrophy of the vessel wall can, however, occur in order to keep up a high blood pressure has been proved by Alexis Carrel[118] and by Fischer and Schmieden,[119] who showed that sections of a vein transplanted into an artery not only did not dilate in consequence of the increased
ssure, but actually became thickened with narrowing
th eir lumina.
abs following schema will be found helpful in contrasting
of whi relating these conditions, any one of which may,
sympto n does, blend with the others :—
tion of ary (Essential or Benign) High Arterial Pressure
those whi ia.
in man. F ury, at times over long periods.
relationship
nitrogen urea, .

B. **Primary (Essential) Arterial Hypertonus, or Hypertonia.**

(*a*) Temporary.

(*b*) Persistent—a "functional arteriosclerosis." Of unknown origin, it may arise independently, and, whether connected with granular kidney or not, is at times associated with excess of erythrocytes in the blood, constituting the condition known as "hypertonia polycythæmica." At first latent, subsequently it tends to cardiac hypertrophy, with its congestive states of headache, irritability, insomnia, and the like. In the large arteries no foci of atheroma are discoverable. Increased blood viscosity is a constant accompaniment of polycythæmia rubra, but neither probably accounts for the rise in arterial pressure which may be due to the onset of congestive heart failure in late stages.

Functional hardness is related to calcification: in both the media is affected, and the condition is general and not focal. Often, however, differential diagnosis is impossible.

C. **Arteriosclerosis.**

(i.) Diffuse hyperplastic sclerosis.
(ii.) Mönckeberg's sclerosis.
(iii.) Senile sclerosis.

D. **Renal States.**—The pathogenetic classification of these is as follows:—

Degenerative—nephrosis.

Inflammatory—nephritis—acute, subacute or chronic.

Chronic nephritis may be:

(i.) Parenchymatous.
(ii.) Interstitial.

(i) *Chronic Nephritis, General or Parenchymatous.*—Glomerulitis and inflammatory new fibrous tissue are distributed throughout the kidneys, with a tendency to association with cardiac hypertrophy and diffuse hyperplastic sclerosis.

Clinically this form is characterised by defective elimination of salt, with resultant production of œdema and ascites.

(ii) *Chronic Interstitial Nephritis.* (*a*) *Partial.*—"The lesion is limited to wedge-shaped areas of fibrosis containing damaged renal parenchyma and more or less small-cell infil-

tration. The intervening tissue is relatively normal, and the parenchyma may be hypertrophied." [89]

True Chronic Interstitial Nephritis. (b) *General.*—There is histological evidence of active inflammation, and the most marked cases of diffuse hyperplastic sclerosis are found in this sub-group

Clinically this form is characterised by defective elimination of waste products of nitrogenous metabolism, which accumulate in the blood, and is associated with cardiac hypertrophy and excessively high and continuous blood pressures.

(c) *True Sclerosis.*—This form may again be subdivided into two, according as the vascular lesion is of the type of—

(α) The arteriolosclerotic kidney.

(β) The arteriosclerotic kidney.

In (α), hyaline material is laid down immediately beneath the endothelium, increasing in amount with progress of the disease. This hyalinisation or sclerosis involves the smaller arterioles of the afferent or efferent vessels, but there is never a true arteritis It is probable that this damage to the arterioles of the kidney is pathognomonic of hyperpiesia.

In (β), there is a diffuse, elastic hyperplasia affecting the intima, and causing thickening thereof, which may extend only as far as the afferent glomerular arterioles. This is the senile form, unless of severe degree at younger ages, when it points to hyperpiesis It is of little clinical import, as it practically never causes renal insufficiency.

So much confusion still exists as to the precise meaning to be attached to the terms hyperpiesis, hyperpiesia, and hypertonia, and these terms are so loosely used by many writers, that the author has deemed it of urgent necessity to clarify, so far as lies in his power, the current obscurity which enwraps the various aspects of high arterial pressure, and to indicate the essential differences between them by means of the above descriptions of each condition and by Table VII.

CHAPTER VII

Arteriosclerosis

"Our age is to be reckoned by the state of our arteries"
Samuel Gee: *Medical Lectures.*

To Lobstein (1833) we owe the term "arteriosclerosis."

Since the publication of Allbutt's well-known views, this complicated problem has been further investigated by an able group of contributors under the editorship of Dr. E. V. Cowdry in "Arteriosclerosis; A Survey of the Problem" (Macmillan, 1933). The following description is a brief synopsis of the work of these and other writers.

Definition

Arteriosclerosis may be looked upon as "a chronic disturbance of the vessels which manifests itself by deposits of the most varied kinds in the vascular walls and which becomes irreversible on reaching its climax in vessels impaired by changes attending the process of ageing with resulting deformation of the lumen and brittleness of the vascular walls" (Aschoff).

Causation of Arteriosclerosis

As to the ætiology of arteriosclerosis, little definite is yet known. In production there appear to be two main elements, (1) changes due to advancing age resulting in distension, dilatation and eventual tortuosity of the arteries along with trophic disturbances resulting in deposition of waste material, (2) pathological changes, which represent the essential nature of the malady.

The various suppositions may now be reviewed in greater detail:—

(*a*) Age changes do not constitute the whole story. Arteriosclerosis in most instances is a senile malady of the

vessels, seldom seen below the age of fifty years and rare above the age of seventy years—the "decrescent" form of Allbutt—independent of known toxin or rise of arterial pressure, but possibly induced by increasing deviation of the acid-base equilibrium to the acid side, a metabolic phenomenon of advancing years, leading to greater difficulties in elimination of waste products.

(*b*) Physico-mechanical effects of wear and tear over long periods of time.

(*c*) Psychical effects of long-continued stress and strain

(*d*) Trophic disturbances.

(*e*) Changes in composition of the blood plasma, and thus of the vascular lymph, leading to absorption, infiltration of vessel walls and precipitation therein.

(*f*) Faulty regulation of metabolism by hormones and vitamins; by unbalanced diets, especially in the direction of prolonged and excessive intake of proteins and lipoids; and by somatic errors of metabolism, as gout, obesity, diabetes.

(*g*) Specific poisons, such as those of syphilis, lead and nicotine; hormonic over-production of pituitrin, adrenaline, thyroxin.

(*h*) Heredity. Osler's [120] references to good or bad quality of the "vital rubber" inherited are well known, and numerous writers [121] [122] have emphasised the part played by heredity in determining vascular disease

(*i*) Habits. Sedentary habits are thought to dispose. Active animals are found to be less liable to arterial changes than those that are more passive.

(*j*) Cosmic factors, such as damp, cold, heat, light climate and atmosphere, have been thought to exert some influence, but evidence as to these is vague.

(*k*) Acute and chronic infections, as well as intestinal toxæmia, have been invoked, but probably have no effect.

(*l*) Alcohol. In the past, alcohol received frequent mention as a cause of arteriosclerosis. The balance of evidence, nevertheless, is entirely against alcohol of itself as an ætiological factor. Out of 283 cases of cirrhosis of the liver associated with excessive alcoholism in persons under fifty years of age, Cabot [123] found that only 6 per cent. had

arteriosclerosis. In another group of 656 arteriosclerotic subjects, only 95 (14·5 per cent.) were under fifty years of age, and of those ninety-five cases only 17 per cent. took alcohol. Mohammedan pilgrims, who never take alcohol, frequently develop arteriosclerosis.[83]

(*m*) Environment. Much discussion has arisen as to the effect of different industrial surroundings on the health of workers. Apart from its special dangers, coal-mining has been as healthy an occupation up to the age of fifty-five years as that of any manual labour. After that age, mortality among miners rises rapidly, mainly because of arteriosclerosis, which was found by Dickson [121] in 91 per cent. of patients, a noteworthy feature being its great frequency in youths of twenty years or under. Arterial pressure was rarely increased. Experts agreed that the physical work done by miners does not exceed that of any class of ordinary labourer. Hence the cause was attributed to the situation of the work, either by reason of lack of sunlight or of the composition of the air breathed. Thus it would appear that sunshine and fresh air are as requisite for middle and later ages as for childhood.

Pathology

The arterial system is composed of three different kinds of vessels: elastic arteries of supply (aorta and its main branches), smaller muscular arteries of various sizes in muscles, organs and tissues (*e.g.*, radial), and smallest arteries (arterioles). Manifestations of reaction to different injuries, though having a broad similarity, differ in individual qualities and degrees.

Probably the intima is first affected. The internal elastic lamina begins to split. This is succeeded by progressive formation of elastic tissue, alterations in the composition of which form the chief feature of the arteriosclerotic process.

The arterial walls may themselves be divided into three layers—intima, media and adventitia. Arteriosclerosis is characterised by involvement of the entire vessel wall, the relative proportions of intimal and medial affection depending solely upon the nature and position of the artery. In

these several layers chemical changes occur resulting in deposition of lipoids, calcium and various proteins. Such chemical changes in the intima—atheromatosis, atherosclerosis, arteriolosclerosis—should be differentiated from calcinosis or calcification of the media, to which the term "arteriosclerosis" is most often limited.

Fibrous tissue and elastic fibres may undergo a further change into hyalinisation. Like changes may occur in muscular arteries, but are then secondary to hypertrophy, and perhaps also to hyperplasia, of the medial muscle.

From this pathological consideration one may proceed to separate out for discussion four main varieties, though, by reason of the many gaps in our present knowledge, neither this nor any other category can be regarded as comprehensive or final :—

I. Diffuse hyperplastic sclerosis, including arteriolosclerosis; atherosclerosis; atheroma.
II. Mönckeberg's sclerosis.
III. Senile arteriosclerosis.
IV. Syphilitic arteritis.

I (A) "Diffuse" Hyperplastic Sclerosis.

Disease of the tunica intima has been variously termed "diffuse hyperplastic sclerosis" (detailed by Jorès,[125] Aschoff,[126] Gaskell[127] and G. Evans[89]), "arteriocapillary fibrosis" (Gull and Sutton),[123] "atherosclerosis" (Marchand, 1904) or in the larger vessels "atheroma."

1. *Morbid Anatomy*.—(*a*) Small arteries and arterioles, especially of the kidneys and spleen, are thickened, and protrude like quills from the cut surface. When arterial pressure has been raised during life, the larger arteries are also thickened and the hypertrophied media appears as transverse ridges on the inner surface of the vessel wall. This is a primary hypertonia, which induces secondary changes in the larger arteries.

(*b*) The heart shows idiopathic hypertrophy, particularly of the left ventricle, a secondary hyperpiesia to overcome a primary hypertonia.

2. *Morbid Histology*.—The essential lesion is a thickening of the intima in the smallest arteries, largely due to intense

tissue activity, evidenced by swelling and proliferation of the endothelial cells, *i.e.*, a primary hypertonia.[200] Secondary fatty degeneration of these cells ensues, leading finally to occlusion of the lumina of the terminal arterioles in the kidney and of the smallest vessels in other organs.

The general distribution of the sclerosis is fairly constant, the kidney being most often affected (a primary hypertonia), and the spleen being the best indicator of the degree and extent of the lesion in the body as a whole. The splenic condition records a secondary hyperpiesia, a second site in which we may estimate the degree of hypertonia, the spleen tissue being soft and reacting readily.

3. *Clinical Associations.*—(*a*) High blood pressure and hyperglycæmia frequently, sometimes obesity

(*b*) Idiopathic cardiac hypertrophy.

(*c*) "It is found in children with renal disease, in adults with chronic diffuse nephritis, and as an incident when death has resulted from some other not related cause." [89]

Christian [129] and Wiseman [130] have drawn attention to the close relation which exists between atherosclerosis, chronic nephritis and certain degenerative changes in the heart, which may be unified as the late stage of an "essential" high pressure condition which began many years before.

Up to, but not after, age thirty, association with chronic nephritis is frequent.

(B) Atheroma.

Atheroma is a patch of "diffuse" hyperplastic sclerosis in the intima of large vessels, but there is no histological difference between patchy and diffuse atherosclerosis, this term well expressing the essential identity of the two varieties.

1. *Morbid Anatomy.*—Distributed haphazard over the aorta and its branches, especially around the orifices of the larger vessels, are nodules of various size which form slightly raised yellowish-white plaques. In the abdominal aorta these tend to be largest and most numerous.

2. *Morbid Histology.*—Similarly to the diffuse hyperplastic form, the essential and initial change is *a thickening of the intima* due to proliferation of the superficial cells and

deposition of lipoids, confined to the intima save where the deeper layers are of loose mesh (aorta, carotids, coronaries) when the media also becomes involved. Later the thickened intima is permeated by elastic fibrils as an essential compensatory change. These lesions may develop into nodular sclerosis or may heal. In certain cases the intimal thickening may be compensatory to a primary affection of the media.

II. Mönckeberg's Sclerosis.

A primary medial calcification, especially favouring the limb vessels, not necessarily associated with intimal changes nor preceded by lipoid change—a primary calcification of the muscle fibres and the surrounding collagen fibres, leading ultimately to necrosis. The process begins in the layer immediately adjacent to the elastic layer, and results in the formation of annular calcareous plaques. There is little or no intimal proliferation, and the not infrequent incidence in comparatively young subjects seems to point to some toxic or infective factor in causation over and above simple wear and tear.

The blood has a potent share in the causation of these vascular affections, for all diseases associated with a large increase in blood cholesterol, such as hypertonia, cardiac disease and diabetes, lead to augmented deposition of lipoid.

For the sake of completeness, one may add to the above—

III. Senile Arteriosclerosis.

1. *Morbid Anatomy.*—A diffuse change, which in established cases is often indistinguishable from I.

2. *Morbid Histology.*—It is *primarily a medial degeneration*, beginning with fatty degeneration and atrophy of the muscle fibres and affecting both larger and smaller vessels (Klotz). This is followed by replacement fibrosis and deposition in the media of lime salts, which, during life, are present in a state similar to that of unset mortar (p. 31), solidifying only after death Intimal thickening, either diffuse, focal or both combined, is generally an accompaniment. It differs from diffuse hyperplastic sclerosis both in respect of site and the characters of its initial lesion.

3. *Clinical Associations.*—In this form cardiac hyper-

trophy and raised blood pressure are both entirely absent. Senile arteriosclerosis is essentially metabolic in character, being probably due to an error in the calcium balance of contractile tissues, but may arise from a potash deficiency caused through lime excess. In other instances it may be primarily toxic. It is doubtful whether purely mechanical factors are responsible for its ætiology. In elderly people cerebral arteriosclerosis may be mistaken for neurasthenia because of symptoms of readily induced mental and physical fatigue, lapses of memory and a lack of mental clarity.[167]

IV. Syphilitic Arteritis.

When recent, this constitutes a distinct type, but later becomes indistinguishable from ordinary arteriosclerosis. The intima is first affected, but all three coats are prone to involvement, and active proliferation, largely perivascular, may be noted in the adventitia.

Hyperpiesis and Arteriosclerosis

Since pathological changes due to arteriosclerosis may occur in childhood, or before the advent of any notable rise in arterial pressure, there is no general agreement as to whether such changes are dependent upon hyperpiesis.

Evidence is accumulating to suggest that hyperpiesis is responsible for arteriolar, but probably not for arterial, thickening. My own view is that intermittent hyperpiesis in the form of angiospasm resulting from sympathetic over-action tends to induce a state of hypertonia, and that hypertonia, maintained over increasingly long periods of time, gives rise to arteriolar constriction and may eventually lead to actual thickening of the arteriolar walls (*vide* Chapter VIII, section 8). On this view arteriolar thickening is secondary and not primary to hyperpiesis.

The position may be thus summarised :—

1. Hyperpiesis can run its course independently of arteriosclerosis.

2. Arteriosclerosis can and does run its course in more than half the total number of cases independently of hyperpiesis.

3. Persistent hyperpiesis *per se* is considered by some not to favour the development of arteriosclerosis.[201] Others are strongly of opinion that it does. At present the matter should be regarded as not finally settled.

Hyperpiesis is independent of Arteriosclerosis

No support can be found for the view, so long held, that the heart has a selective action which enables it to overcome a general or local resistance due to arterial occlusion [131] [132] nor for the deduction that hyperpiesis is consequent upon arteriosclerosis for which age changes have been held responsible, in view of the fact that sphygmomanometric findings have established both the presence of hyperpiesis in the absence of any factors which induce arterial narrowing and, conversely, the absence of hyperpiesis in healthy and vigorous old men, whose pressures remain constantly below 150 mm Hg. Indeed, it may now be taken as amply proved that arteriosclerosis without high arterial pressure is a clinical and pathological entity.[133, 134, 135]

Hyperpiesis and Atheroma

The causal relation between hyperpiesis and atheroma is supported by considerable evidence. The readiness with which blood serum penetrates the walls of arteries is highly significant, and this depends both upon their structure and upon the height of the arterial pressure. Certain diets are known to produce atheroma, which leads to the hope that a diet capable of staving off the onset of this disease may eventually be discovered.

CHAPTER VIII

The Causation and Significance of High Arterial Pressure

"High pressure is an attempt of the organism to maintain the equilibrium of its circulation" Allbutt. *Diseases of Arteries.*

The primary cause or causes of pathologically high arterial pressures are not yet definitely established.

Hence this fundamental difficulty confronts us as soon as we attempt to get back to first principles. Much confusion, too, exists by reason of the many different names employed by numerous authors to describe conditions which are identical or overlapping, some attacking the problem from biological and clinical standpoints, others from a groundwork of morbid anatomy and pathology.

While we are tentatively assured of certain factors being associated with the origin of hyperpiesis, we are unable to point to any one with a definite conviction of being right. Yet the time is probably not far distant when the real causes will be brought to light, and measures taken that will prevent the development of conditions which to-day are responsible for a mortality that is far too high.

Transitory variations from physiological standards for age and weight capable of wide modification to meet the varying needs of daily life have been dealt with in Chapter V. We now have to consider temporary or permanent variations within still wider ranges due to some underlying cause or *causes of pathological origin.*

CAUSAL FACTORS

In the light of our present knowledge the following considerations lend support to a provisional attempt to tabulate the causes of raised arterial pressure as follow :—

1. Compensatory.

2. Mechanical—the effect of "wear and tear" upon the cardio-vascular system.
3. Hereditary and familial influences.
4. Arteriolar constriction.
5. Metabolic influences.
6. Deficient elimination.
7. Toxæmic influences. (*a*) Bacterial, (*b*) chemical.
8. Psychical influences.
9. Endocrino-sympathetic influences.
10. Allergy.
11. From the interaction of two or more of the above causes.

1. **Compensatory Influences** to maintain an efficient circulation.

2. **Mechanical Influences.**—It has been suggested that in hyperpiesis associated with arterial degeneration, the effects are due to "wear and tear" upon the cardio-vascular system, the initial qualities of the "vital rubber" conditioning the degree of arterial resistance to stresses and strains.

The main mechanical cause of high arterial pressures is increased peripheral resistance, *i. e.*, "an augmented frictional resistance to the onward passage of blood from the arteries, dependent on narrowing of the outlet by active contraction of the arterioles or by structural change" [67] (*vide* 4, *infra*)

3. **Hereditary and Familial Influences.**—These undoubtedly play a part, as is seen in cases of congenital narrowing of the blood vessels, and in those which tend to early arterial degeneration by reason of the poor quality of elastic tissue of which their walls are composed. Again, the quality may vary in different branches of the arterial tree. A family tendency is detectable in more than one-third of all histories.[137]

4. **Arteriolar Constriction.**—Formerly it was believed that, with a constant cardiac output, the amount of blood flowing through the capillary circulation was regulated primarily by the arterioles from which they originate, and secondarily by the venous pressure; in other words, that passive dilatation of the capillaries with rise in intracapillary pressure occurred when the arterioles dilated, while, when

these latter contracted, the capillaries by reason of their elasticity resumed their state of tonic contraction.

As regards ætiology, diagnosis and treatment, however, in 1919 Krogh [138] opened up a new and valuable line of thought by bringing forward evidence, which has been corroborated by Lewis,[139] Dale [140] and other observers,[141, 142] that the arterial and capillary systems are regulated by their own special mechanisms, and thus react and function independently. Krogh has proved that not only do the capillaries contract and dilate apart from the influence of the arterioles, but that their calibre when the tissue they supply is at rest is considerably less than when the tissue is active, showing that the capillaries exist in a state of slight constrictor tone.

Two Probable Groups of High Arterial Pressure.—A. Without high capillary pressures.

That active constriction of the arterioles precedes the onset of œdema and of hæmorrhage into the retina can be demonstrated most readily in cases of toxæmia of pregnancy. From this and other evidence it seems obvious that spastic constriction of the arterioles is a significant factor in the causation of hyperpiesis itself and of many of the symptoms and complications. Experimental results further show that in renal arteriosclerosis the arterioles alone are involved, the capillary pressure remaining normal.

B. With high capillary pressures. In chronic interstitial nephritis the capillary system is associated with, even if not the actual cause of, the raised pressure. It is probable that the more severe and rapidly progressive types of hyperpiesis have their earliest manifestations in a diffuse lesion of the vascular system (diffuse hyperplastic sclerosis) of which involvement of the kidney is only a part, and not, as *previously supposed, the cause.*

In contrast to extensive fluctuations which occur in states of simple high arterial pressure, the pressure in the capillaries is usually normal or even below normal. Thus, a systemic pressure of 200 mm. in the brachial artery becomes broken down to one of 20 mm. in the capillaries of the fingers with corresponding obliteration of the differential pressure.[143] This damping down of pressure must necessarily take place

in the arterioles, which act as transformers, and has a greater effect in regulating capillary circulation than the arterial pressure itself.[144] It is the resistance in the terminal vascular bed which not only reduces high to low pressure but converts the intermittent, pulsating arterial flow into an even, continuous capillary current. Hence the real seat of rise in systemic arterial pressure lies in increased tone of the tiny arterioles, their smooth muscle cells being the anatomical tissues directly concerned.

Since the blood mass cannot fill more than half the vascular bed, and since local vasoconstriction is readily balanced by vasodilatation elsewhere, hyperpiesis "can only arise when the machinery of regulation is damaged or the pressor stimuli are too powerful and overcome the depressor mechanism" [131]

For such elevation of pressure to be induced, increase in tone of the muscle elements of the arterioles must be widespread Constriction limited to the vessels of the kidney, to the splanchnic area or even to the extremities is insufficient to bring about hyperpiesis [145]

"In the healthy person at rest the arterial pressure which is normal for that individual is remarkably constant within narrow limits, apart from ascertainable disturbing conditions; the regulating nervous mechanism (medullary centres) is very efficient. But in the high-pressure subject this regulating mechanism has ceased to be adequate; the pressure remains at an abnormally high level, and is often subject to sudden and extensive variations, sometimes in the absence of any recognisable cause or from slight exciting influences that would have little effect in the normal individual. The normal regulating mechanism includes the depressing or inhibitory effect on the vasomotor centre of high blood pressure acting (1) directly on the centre, and (2) through afferent impulses from the heart, aorta, sinus caroticus, etc. In addition, there is the controlling influence of the vagus centre on the cardiac pump, this centre being also influenced directly by the blood pressure in it and reflexly by afferent impulses from the heart, aorta and sinus caroticus." [67]

What, then, are the underlying causes that in high

pressure states bring about either temporary arteriolar spasm or more permanent vasoconstriction ?

5. **Metabolic Influences.**—The precise nature of the neuro-humoral impulse that primarily induces neuro-hypophysial basophilia, and thus secondarily activates the vasomotor centre in the medulla, has yet to be determined. It may be that ischæmia of the centre is brought about by a selective constrictor effect upon its arterioles exerted by an autogenous toxic substance, itself arising as a result of disordered metabolism.

Up to now no one chemical substance circulating in the blood can be invoked as the primary cause. From the knowledge afforded by recent biochemical investigations into the chemistry of the body, it would not be difficult to assume that all hyperpiesis begins as a disturbance, whether as a result of toxæmia or otherwise, of the body metabolism, a tissue-resistance problem involving cellular, lymphatic and arteriolo-capillary balance which varies enormously with age and with deviation of the acid-base balance in the direction either of acidity or alkalinity.

6. **Deficient Elimination.**—Deficient elimination through the kidneys, skin and lungs is another important factor, since the body becomes thus unable to get rid of its waste material.

Attempts have also been made to show that, apart from the rise in blood pressure associated with efforts on the part of the kidneys to secure effective elimination of concentrated urine, salt or protein, heightened blood pressure may be due to endeavours of the kidney to secure adequate elimination of acid waste products when their production is in excess of normal, or when there is some defect, functional or structural, in the renal mechanism. Much further evidence on the association of acidosis with high blood pressure is, however, still required.

7. **Toxæmic Influences.**—In the cases with normal kidneys some prerenal causal factor must be sought for hyperpiesis, which is often afforded by *chronic toxæmia*, induced by :—

(*a*) *Bacterial Intoxication.*—This arises from septic foci, *e.g.*, tonsils, gums, teeth, gall bladder, appendix, etc. The intestine is the main source, Bacillus coli being largely

responsible. Streptoccal or pneumococcal infection of the upper air passages or lungs causes destruction of red blood cells with resultant lessening of processes of oxidation in the blood. B. coli may then become deprived of its free oxygen, and so have to alter its mode of living. Organisms swallowed in the saliva also find their way into the intestine, so that the latter becomes a nidus for organisms which begin their activities in the mouth or respiratory system. These can usually be isolated from the fæces. After some time the liver becomes unable to carry on detoxication adequately enough to rid the body of abnormal products of metabolism set up by these organisms, the evidence being a slow rise and fall in the blood sugar tolerance curve, the opposite of the normal picture. It is probable that direct effects upon the blood vessels may be caused by bacterial toxins in like manner with syphilis, or that indirect influences may be exerted through damage to the kidney during excretion of live bacteria.

Under the influence of infections proteinogenous substances may be formed in the blood, capable of inducing spasm, similar to those which culminate in the eclampsia of pregnancy. Under ordinary circumstances they are dealt with by the liver, but when the liver suffers as the result of infection, these substances accumulate in the blood, and high blood pressure results from spasm of the arterioles. The effects of toxæmia are most apparent in the kidneys by reason of their vital importance, the manifestations produced thus forming only one symptom of the general poisoning.[146]

(b) *Chemical Intoxication.*—(i) Arising within the body. Digestive troubles of the nature of spasm and atony of stomach and bowels with impaired secretion of digestive juices may arise, as a result of sympathetic nervous dysfunction. Some of these toxins have been identified; others are only surmised. They are capable of producing states of high and low pressure, and originate most frequently in the course of intestinal stasis with resultant fermentation, putrefaction and absorption of poisonous products of incomplete digestion into the blood stream. Putrefaction arises most often from a high protein diet, split products of protein digestion being formed through faulty metabolism, thus giving rise to

chemical poisoning which appears to cause endocrine stimulation sufficient in amount to produce irritability of the vasomotor system leading to initial transitory changes in arterial pressure, which later become permanent.

Biochemistry has shown that chemical bodies of the nature of amine bases are formed during putrefaction, *e.g.*, skatol, and even during prolonged peptic digestion, the various symptoms depending upon pathological absorption from the gastro-intestinal tract of different amine bodies, some of which are pressor whilst others are not. "Designed as an intensive preparation for action or defence, the sympathetic response may be so dissociated, perverted or prolonged as to disorganise digestion by exciting spasm and atony in stomach and bowels and inhibiting the secretion of digestive juices; it may keep blood pressure at a level which is inappropriate for the task of the heart and the arteries. These effects are not necessarily distinct. Thus intestinal stasis from sympathetic inhibition causes poisons of putrefactive origin to be absorbed, which in their turn lead to vasoconstriction, and hence an unduly raised blood pressure." [60] According to Robertson,[147] the aliphatic monamines exert a physiological action akin to that produced by stimulation of the sympathetic nervous system, the most active of the monamines derived from the amino-acid cleavage products of protein being tyramine, which on intravenous injection causes a rapid and pronounced rise in arterial pressure about one-twentieth of that exerted by adrenaline but more prolonged.

The proof of the existence of intestinal auto-toxæmia is the presence in the urine of indican, when constant and in undue amount, but in hyperpietic states this is seldom found, and injections of indol are without effect upon arterial pressures. At the same time, it is probable that arterial degeneration in various kinds and degrees results from absorption into the blood stream of certain toxic substances. The respiratory tract is also another source of chronic toxæmia.

Hence, although we cannot point to any one factor as the definite source of origin of supernormal arterial pressure—and it is highly probable that more than one source exists—

are the opposite of the child. They do not play. They have no illusions. They are tense and irritable, with 'single-track' minds. While their mental horizon is narrow, within this range they are terribly concentrated, and pursue their aims with grim desperation.

"Many belong to the class of the successful, if by 'success' one refers to accumulation of wealth or power that is not always accompanied by a spiritual or ethical uplift. One can readily grasp why such individuals are successful, for they throw everything in life aside, especially play, that does not directly contribute to their purpose One of the commonest and most pathetic experiences in medical practice is the tragedy of the 'successful' man.

"On the other hand, it is not always the desire for 'success' that causes mental conflict. Those who have fallen by the wayside, the poor and the meek, acquire hypertension as well. Most have lived what may be called a 'hard' life. The struggle for existence has begun early, and their life represents little else than a desperate battle.

"They have no time for play. Here again the child that is in all of us goes early, and once gone rarely heeds the summons to return.

"There is no question in my mind that such constitutions are acquired and not inborn. One sees occasionally hypertensive disease and its consequences common in certain families. This is the result of the imitative tendency of children."

The above graphic picture, though highly coloured, delineates a characteristic sub-group of hyperpiesis. Two of this author's points, however, are open to question. The first is that which relegates such type of constitution entirely to acquired characteristics, and ignores the effects of heredity. The second is that which assigns to a certain fixity of mental processes a corresponding bodily form. In this country at any rate one sees hyperpiesis of psychical (or acquired) origin in the tall as well as in the short; in the nervous, thin and emaciated as well as in the obese. Nevertheless, some cases, including "captains of industry," do fall within the limits described by Moschcowitz, but I have found the well-rounded contours of such men commonly to

be due to physical causes such as "business lunches," public dinners, big cigars, luxurious motor cars and sedentary occupations rather than solely to a tense mental attitude towards life. Certain it is that in the United States the busy public man lives continuously at extreme high pressure, as I have witnessed, and is quite unable to relax even when playing games. He is out to win every time, and concentrates every faculty on so doing. May we venture to think that our more sport-loving countrymen are wiser in their generation in that they appreciate more fully the advantages of leisure, and are not for ever sitting upon the safety-valve?

Further research is required as to the biochemical and electrical changes which nerve cells undergo during activity. As to these, we know that wear and tear of nerve cells causes disintegration of the complex protein and albuminoid substances of which they are composed, and we are justified in drawing an analogy between these and the pressor effects due to the breaking down of protein bodies by bacteria. Sudden shocks, mental emotion and excitement are all potent in raising the arterial pressure for the time being to extraordinary heights, whilst continued worry and anxiety conditions have a strong action in promoting vasoconstriction over long periods of time, tending ultimately to permanent thickening of the vessel walls.

Of the somatic neuroses, in general terms we may say that in true anxiety neurosis the blood pressure is often raised, whilst in neurasthenia it is usually low. Pseudo-neurasthenia, nevertheless, presents the symptom complex of neurasthenia, but these symptoms actually originate from hyperpiesis, with or without arteriosclerosis, gastric disturbance or chronic nephritis, and the heightened blood pressure will put us on the right track. I have seen in consultation patients over sixty in whom the presence of cancer was suspected by the local practitioner by reason of the gradual pallor and loss of weight, these symptoms being in reality due to arteriosclerosis.

9. **Endocrine-sympathetic Influences.**—The importance of the sympathetic endocrine group is becoming increasingly recognised. "As both endocrine glands and the sympathetic nervous system became specialised they remained associated.

This association is reciprocal, as not only does the sympathetic nervous system stimulate the secretion of these ductless glands, but their secretion increases in turn the sympathetic response. Thus the sympathetic nervous system, the endocrine glands, and the gonads form a basic tripod, whose relationship is shown in disease as well as in health, and is reflected in many of the neuroses and psychoses." [60]

This brings us to the question as to whether hyperpiesis in its early course is a disturbance of function which leads to changes in structure, or whether it is preceded and produced by changes in structure.

If one desires to study the beginnings of hyperpiesis, it is necessary to do so before the onset of complications. The solution of this problem has been sought through investigations of apparently healthy schoolchildren and adolescents.[136] Results of such study go to prove that hyperpiesis begins as a transient functional phenomenon in subjects with an over-responsive vasomotor system when exposed to physical or psychical stress. "Commonly the blood pressure at rest is above normal, but not always. It might be even a family trait. Usually the activating cause is apprehension, and anxiety for success, the link between the higher centres and the vasomotor centre being too intimate. . . . What is in store depends on circumstances; whether the temperament is placid, or eager and over-anxious; and on the nature of the employment. Arterial pressure might remain within the normal until the responsibilities of maturity and the metabolic imperfections of middle life reinforced the tendency." [149] In later life it may be a reaction to the hurry and strain of modern civilisation in the form of a heightened excitability of the vasomotor centre. The chief ductless glands implicated are the pituitary and adrenals (which increase the sensitiveness of response to the sympathetic system under the influence of emotion or of infection), and to a less extent the thyroid gland and gonads.

Cases of heightened arterial pressure associated with hyperthyroidism, adrenal and pituitary over-activity, usually with some evidence of hypertrophy are not uncommon, and, when recognised, adequately explain the rise in pressure and suggest appropriate treatment.

10. **Allergy.**—Bishop[150] has advanced a simple yet wide conception of a general cellular disorder from which few cells of the body are exempt. The heart, blood vessels, liver and kidneys suffer conspicuously because of their importance and activity, and because of the extra strain to which they are subjected by cellular damage. "Cardio-arteriosclerosis" would thus include hardening of the heart and arteries, progressive cardiac failure, Bright's disease and presenility. It is suggested that a reactive irritation is set up against some material with which the cells have to deal in the processes of cellular nutrition, and that this irritation, which Bishop regards as allergic in nature, leads to cell injury with ultimate destruction and replacement by fibrous tissue. Manifestations of this take place in various organs, *e g.*, in the eye, causing neuroretinitis, in the kidney, causing nephrosclerosis, etc.

The extraneous material is believed to be a protein or protein derivative from ordinary food, or from the bodies of bacteria to which the cells of the patient have become sensitive. Such sensitiveness dates usually from some acute illness of the nature of gall-stones, appendicitis, typhoid, malaria, etc., or from shock or great mental perturbation, or again from acute food poisoning. Strickland Goodall,[151] who examined 2,000 cases under the age of forty, found that the most frequent antecedent of hyperpiesis was scarlet fever. These findings favour the infective origin of certain forms of hyperpiesis and support the view that hyperplastic sclerosis of the intima is the underlying change.

Allergy has been invoked also by Waldbott as a cause of hyperpiesia, but Cohen, Fineberg and Rudolph are in opposition to these conclusions.

Hyperpiesis is not necessarily a Symptom of Cardio-arterio-renal Changes

It used to be thought that persistently increased arterial pressure was in every case symptomatic of vascular, cardiac or renal changes, each bearing its own individual label, as, for example, thickening of arterial walls, cardiac hypertrophy

and impaired renal permeability, these manifestations appearing jointly or severally with individual variations in kind or degree.

One fact is certain, namely, that raised arterial pressure, transient, intermittent or even permanent, sometimes also of high grade, frequently manifests itself in the absence of any evidence of kidney disease.

"That patients with essential hypertension may later in life develop cardiorenal complications affords no justification for the conclusion that the primary cause of the high blood pressure lies in the organ or organs which secondarily show evidence of disease. Study of histories of patients and careful continuous clinical observation will reveal many patients with hypertension of fairly high grade and yet with no evidence of myocardial degeneration, arterial change or disturbance in kidney function."[152]

Cardiac decompensation with marked passive hyperæmia of the kidneys is associated with a moderate retention of non-protein nitrogen in the blood, and the height of the blood pressure bears no relationship to the amount of the non-protein nitrogen substances in the blood. High arterial pressure with normal non-protein nitrogen values in the blood and normal kidney excretion, determined by functional tests and urine examinations, does not justify the clinical diagnosis of chronic nephritis.

In certain cases the rise in pressure is of the nature of a compensatory and protective mechanism induced by factors which have for their object the maintenance of an efficient circulation through the various organs and tissues.

Association of Hyperpiesis with Renal and Myocardial Involvement

Wallgren[153] examined kidneys from forty-four patients with high blood pressure, comparing them with fifty-one controls with normal kidneys. Eleven out of the forty-four cases (hyperpietic) exhibited blood vessels conforming with those in the normal age group. In the remaining thirty-four cases (hypertonic) typical nephrosclerosis was found, the condition of the blood vessels differing quantitatively, but

not qualitatively, from the changes of normal development and ageing.

Clinical and *post-mortem* diagnoses of nephritis do not always tally, and the presence and degree of high pressure bear no absolute relation to the amount of kidney involvement. In fact, one or the other may be entirely absent.[154, 155] Although there are still certain observers, including Lian and Haguenau,[156] who affirm on clinical grounds that acute or chronic nephritis is, in certain cases, the exciting cause of a transitory or permanent rise in arterial pressure, nevertheless cases are recorded by Moschcowitz [148] in which raised arterial pressure and clinical evidences of nephritis were present, although the kidneys showed but slight lesions. His experimental and clinical observations tend strongly to prove that even in cases of definite nephritis high arterial pressure may be the earliest demonstrable symptom, and lend no support to the belief that, when associated with nephritis, it is of renal origin. Rather does he incline to the idea that arteriocapillary fibrosis is merely the localised and prominent manifestation of a generalised vascular affection, which explains the frequency of clinical phenomena referable to other organs, *e.g.*, brain, aorta, heart, pancreas, arteries, etc. On this view arteriocapillary fibrosis (diffuse hyperplastic sclerosis) and arterial disease are contemporaneous reactions to the same hurt. In other words, a hypertonia produces a secondary hyperpiesis.

The end results of a glomerular nephritis with secondary contracted kidney and of a primary contracted kidney are morphologically the same. The causes must be identical. In the former there is a blood pressure which exceeds the normal; in the latter vascular lesions occur even at times with normal pressures.

"Diffuse hyperplastic sclerosis shares with chronic nephritis and chronic interstitial nephritis a common ætiology, which, so far as is known at present, is due to the action of bacterial toxins, and both the character of the vascular lesion in diffuse hyperplastic sclerosis and the distribution of the lesion in the vascular tree are compatible with the view that it also is caused by a circulating toxin." [88]

Such toxins, however, probably do not act unless an error

of metabolism is present as the fundamental basis producing a condition of hypertonia which may induce a secondary hyperpiesis.

It is in accordance with numerous observations that cases of renal affection are met with in which hyperpiesis, either transitory or permanent, coincides with the bacterial infection or with the intoxication which induces a process of acute or chronic nephritis.

Hence we should rightly regard the kidney lesions in nephrosclerosis as due to injury of the vessels, which arises from the cause or causes which simultaneously produce the high pressure. Moreover, the effects upon the kidney will vary according as to whether arterioles or capillaries are the more directly concerned.

Like causative factors are operative in the production of myocarditis, and in all three conditions—raised blood pressure, nephritis and myocarditis—disturbance in the arterioles constitutes an integral part of the reaction to injury. Rise in blood pressure and œdema are thus earlier indications of kidney affection than protein, casts and erythrocytes in the urine. So, too, certain causal factors give rise to the appearance of œdema from disorganised capillary mechanism akin to its manifestation in stasis from cardiac decompensation.

CHAPTER IX

Symptomatology of High Arterial Pressure

"Supertension has no rigid limits or defined boundaries, and passes gradually, perhaps silently, into states of disease, as manifested, for example, by damaged arteries or defeated hearts."

Lord Dawson of Penn: *Proc. Roy. Soc. Med.*, June, 1926.

A. Symptoms of High Arterial Pressure

In its earlier stages, and over a period of time varying with the individual, apart from transient bleeding, usually from the nose, simple and uncomplicated high arterial pressure has no symptoms. Indeed, the subjects are often conscious of no disability, and feel particularly hale and hearty. Many of them up to middle life exhibit a high degree of mental and physical activity, the condition being discovered only on medical examination for life assurance or for some minor symptoms for which they seek advice. Their metabolic processes are usually in excess, but of this they are blissfully unconscious.

From this pre-sclerotic phase they gradually pass into a state in which vague symptoms and signs afford an often-disregarded warning.

The symptoms of hyperpiesis may be any one or more of the following, most of which are due to disorder of the circulatory mechanism in which the central nervous system is involved, certain of them constituting evidences of congestion, cerebral or otherwise, the result of overfilled blood vessels.

The earliest and most frequent subjective disorder of sensation of which the patient complains is frontal or vertico-occipital *headache*, commonly dull, and aggravated by attempts at mental concentration, less often acute and throbbing. Occasionally headache is associated with *dizziness* on rapid change of position, as in rising after stooping or getting up suddenly from the recumbent posture.

True vertigo is rare. Sensations of *fullness or heaviness in the head* may be experienced. A symptom often more obvious to relatives and friends than to the patient is a *change in temperament.* From being calm and equable, evidences of lack of control become apparent in the shape of *irritability* on slight or no provocation, with sudden and ungovernable fits of anger. *Drowsiness and disinclination* for effort cause social, business and domestic duties to become irksome and neglected. Nervous symptoms, with or without *emotional outbreaks,* may occur, particularly in women, in whom they tend to become more pronounced at or after the menopause *Memory is increasingly impaired,* along with *incapacity for sustained mental or physical effort,* eventually leading to a state of complete *exhaustion.* Anxiety neurosis with vague apprehensions of impending disaster, *neuralgia, migraine* and *mild psychic exaltation with confusion of ideas* may also supervene. With high pressures arterial pulsation is often perceptible as a throbbing sensation when recumbent, and sometimes prevents sleep.

In established cases the symptoms attributable to hyperpiesis proper begin to be interwoven with those of the precedent, induced or associated morbid conditions. Thus the sallow complexion and loss of weight of certain advanced arteriosclerotic subjects in conjunction with nervous depression, exhaustion and symptoms referred to the gastro-intestinal tract may cause the condition to be mistaken for neurasthenia unless the arterial pressure be taken. In over 50 per cent. of established cases of hyperpiesis, palpitation, flushing, precordial pain and dyspnœa are apt to occur in the absence of primary myocardial or valvular disease, generalised arteriosclerosis, renal disease or hyperthyroidism. Retinal hæmorrhages may be present in about 5 per cent. of all cases. A rude awakening may happen from one of the accidents consequent upon the increasing pressure-load, and with fully developed sclerosis of heart, arteries and kidneys still more dramatic issues impend and may come to pass, often with fatal results.

The high-pressure individual is often the subject of excess metabolism and labours under the strain of acid retention, being unable adequately to eliminate waste products. The

acid-base balance of the tissues is deviated to the acid side, and tissue excess formation, particularly of fat, preponderates over destruction and elimination of waste products.

B. Signs of High Arterial Pressure

The single sign of uncomplicated hyperpiesis is the height of the arterial pressure recorded by the sphygmomanometer.

Effects of Persistently Raised Arterial Pressure on the Heart and Arteries

Protracted high arterial pressure causes the heart to enlarge. If, therefore, in a case of high arterial pressure the heart is found to be of normal size, one may conclude that the heightened pressure has not been of long duration. A persistently raised pressure is bound to lead to general cardio-vascular hypertrophy, with sclerotic changes, especially in the middle coats of muscular arteries and in the left ventricle of the heart. So long as the left ventricle successfully overcomes the resistance by adequate systole, progressive hypertrophy only occurs. Directly it fails to do so, and an excess of residual blood remains after systole, dilatation gradually ensues with degeneration of the muscular tissue. Whether the arteries dilate or not under a persistently raised arterial pressure depends upon their tone. The radial arteries, for example, may have sufficient contractile power to resist heightened pressures for years without becoming dilated and tortuous. Similarly with the aorta, though the brachial arteries tend to become involved at an earlier date. Contracted muscular tissue, while still unimpaired, is elastic and resilient, arterial muscle thus permitting the arteries to yield and to recoil

C. Effects of High Arterial Pressure

1. *On the Heart.*—Even over long periods it is astonishing to find in many cases little induced change in the heart, this organ being influenced solely by increased load (hyperacthia). Palpitation and tachycardia may be due to forcible cardiac action of nervous origin, or to extrasystoles. In the majority

of established cases, however, the heart undergoes hypertrophy as a result of its overload, manifested clinically by augmented heart rate and lessened response to effort. The slowly progressive left ventricular preponderance can be confirmed by percussion, X-rays and the electrocardiograph.

Subsequently dilatation supervenes, with blowing systolic murmurs, mitral and sometimes aortic, the latter constituting "the cardio-aortic type of hyperpietic heart" described by Vaquez. Myocardial degeneration leads to cardiac failure, of which increasing breathlessness on exertion is a frequent symptom. More advanced cases may suffer from distressing nocturnal attacks of dyspnœa, the so-called "cardiac asthma," with or without Cheyne-Stokes breathing.

The rule is for myocardial failure, whether or not the result of cardiac defeat, to be accompanied by distinctly low pressures, but, every now and again, one comes across cases of arterial pressure which have overtopped the 200 mm. mark, associated with a failing heart and feeble pulse. Such cases present many points of interest. "The first explanation that occurs is that the heart is failing behind the high pressure, but if so, lowering the pressure should relieve the heart, which, as a matter of fact, it fails to do. The next explanation offered was that as the output of the heart diminishes, vasoconstriction must occur to diminish the size of the bed to be filled, and thus to spare the heart. This would account for pressure being maintained at its normal level, but not for its rising as the heart fails. But we now know that the strongest stimulus to a muscular contraction is the previous stretching.[199] Hence the marked hypertrophy of the left ventricle in aortic regurgitation, where the ventricle is filled during diastole, both from the auricle and from the aorta, and so is stimulated to increased work. In the same way we may regard the vasoconstriction produced through the sympathetic as an attempt, by raising blood pressure, to stimulate the flagging heart, for the diastolic stretching will be increased thereby. But when the myocardium is diseased it fails to respond to a remedy which is at best a desperate one—the overstretched muscle fails to respond and dilatation increases." [60]

2. *On the Arteries and Arterioles.*—These vessels may be

CHAPTER X

Diagnosis and Prognosis of High Arterial Pressure

"If I were allowed to have only two instruments of precision for my aid in physical diagnosis, they would be the stethoscope and the blood pressure machine."—Cabot: *Lectures.*

A. DIAGNOSIS OF HIGH ARTERIAL PRESSURE

Clinical Examination of the Patient.—For accurate diagnosis of high pressure conditions, examination of the patient cannot be too careful. I recommend the following order as being the most convenient :—

1. History.—This must be taken carefully, special attention being directed to :—

(*a*) Family history of gout, rheumatism, syphilis or alcohol.

(*b*) Personal history of antecedent infections, especially nephritis and syphilis ; occupations involving contact with lead or involving physical and mental strain ; emotional shocks ; long-continued worry ; too little sleep ; wrong habits, particularly as regards over-eating, errors in diet, hurried meals, abuse of alcohol or tobacco ; habitual constipation ; chronic sepsis, focal or general.

2. Systematic Examination of the Cardiovascular System.—(*a*) Take several readings from both brachial arteries and discard the first. Record the *pulse rate and characteristics*

(*b*) Note *position of apex beat, nature of cardiac sounds* at apex and both bases, *always with the chest bared, and state of superficial arteries.*

3. General Condition of Patient.—Inspect for presence or absence of œdema. It should be remembered that hydrostatic dropsy is of frequent occurrence in patients over middle age, of sedentary habit and heavy build, and does not imply cardiac failure unless attended by increased general venous pressure. Such rise can readily be determined by gradually

the polynuclear cells is on the whole smaller than normal. The test should be performed by a trained histologist.

C. BIOCHEMICAL INVESTIGATIONS

Tests of Renal Efficiency.—Where disturbance of nitrogenous metabolism is suspected, recourse should be had to biochemical tests, which are divided into those performed on the urine and blood respectively.

By the former we learn what the patient is excreting; the latter tell us what he is retaining.

As regards clinical utility, the latter factor is of much greater importance than the former.

(a) Urinary Tests.—In regard to urinary tests, it must be emphasised that simple estimation of the amount of urea present in the urine, even in a twenty-four hours' specimen, is of little value; in a "casual" specimen, it is not only useless but misleading.

Many elaborations of urinary methods, such as the urea concentration test and its derivatives (Maclean)[158] and the water elimination tests (Calvert),[159] have been employed, but are all open to the practical objection that in any given case they may mislead, for it is now recognised that at times a person with disorganised kidneys may be found to concentrate urea within normal limits, while, on the other hand, persons in whom no clinical kidney defect is apparent fail to concentrate urea sufficiently.

(b) Blood Tests.—"Retention" tests on the blood are now regarded as essential, and should be performed whenever the functioning power of the kidneys is in doubt, as there is no purely clinical method of determining this most important factor.

(α) *The Blood Urea Test.*—The test is performed as follows: not less than 3 c.c. of intravenous blood are withdrawn into a tube coated internally with sodium fluoride. This is treated with urease (a ferment derived from the Soya bean) in the presence of a buffered phosphate solution, which maintains a suitable *p*H in the mixture. The ammonia is then liberated by strong alkali, and is either distilled or aspirated by a current of air into standard acid solution and finally estimated either by

titration against standard alkali or colorimetrically after the addition of Nessler's reagent.

A blood urea content of 40 mgms. per 100 c.c. or under is regarded as normal. In the interpretation of figures above this, it should be remembered that in acute intestinal obstruction high readings may be obtained which are not necessarily indicative of a failing kidney. With this exception, a reading above 50 mgms. should be regarded as conclusive of some degree of nitrogen retention and as a factor in the causation or maintenance of a high blood pressure.

(β) *Blood Calcium and Blood Phosphorus Estimations.*—In any case of hyperpiesis it is always of help to estimate the blood calcium and blood phosphorus content. In regard to calcium, what is desirable to know is the degree of calcium retention, the degree of calcium excretion is relatively unimportant In a number of cases of hyperpiesia the blood calcium figure is found to be low. On the other hand, in many cases of hyperpiesis with which arteriosclerosis is associated the blood calcium figure is found to be high (*vide* p. 123).

D. PROGNOSIS OF HIGH ARTERIAL PRESSURE

On accurate diagnosis correct prognosis and treatment alike depend.

In any consideration of prognosis in high arterial pressure, one must take into account the state of the heart, of the arteries and of the kidneys

The first step is to exclude the presence of syphilis. The next step is to differentiate hyperpiesia from hyperpiesis and hypertonia due to chronic renal disease. In hyperpiesia the prognosis is better than in chronic renal disease, more especially when coming under observation in its earlier stages. When associated with obesity the prognosis in the majority of cases is good.

Determine by repeated tests whether a rise in pressure is temporary or permanent. The systolic transitory type, due to emotion or excitement, is commoner in women than in men, and may be diagnosed by the appearance of the patient and the absence of cardiac hypertrophy, arterio-

sclerosis, proteinuria, retinal changes and other manifestations which accompany the fully developed condition. Part of the rise may be due to arterial spasm. If uncertainty exists, rest for a few days in bed clears up the diagnosis by reducing the unstable nervous pressures to within normal limits.

Favourable Features.—The outlook is best where removal of severe mental stress brings about a cure, or where the condition is revealed fortuitously, as during examination for life assurance, and the patient is leading a full life without symptoms, effort-tolerance being good. With appearance and increase of circulatory or renal defects, the prognosis correspondingly becomes worse. Maintenance of a quiet mode of life, with abandonment of strenuous efforts, frequently brings about prolonged improvement. For this reason the prognosis is better in elderly women than in men of equivalent age.

Unfavourable features are increased pressure at comparatively early ages—the younger the patient, the worse the outlook; family history of vascular disease; a relatively high diastolic pressure; evidences of systemic degeneration in arteries, heart, kidneys and digestive tract, limited effort-tolerance.[160]

Very high pressures are always unfavourable, but pressures of moderate grade have to be considered on their individual merits, and may be requisite for maintenance of the visceral pulse. On the whole, prognosis is far less grave than formerly thought.

Finally, since prognosis largely depends upon whether high arterial pressure is associated or not with clinical evidences of chronic nephritis, before expressing any opinion it is wise to watch the effect of treatment, and to try daily catharsis with sodium and magnesium sulphates. Failure to react to treatment of this kind is practically pathognomonic of the incidence of renal disease.[160]

"In many persons, even in the quite elderly, phases of high pressure occur which prove to be transient; though probably recurrent. Much will depend on the biochemical reports on the blood and the urine, and on the course of diastolic pressures; diastolic pressures round about 100 are

of ill-omen. Response to treatment, on the other hand, is a favourable sign. The peril lies in the bigger incidents; in cardiac defect, or apoplexy, or pulmonary œdema. If, however, the high pressures prove to have a renal origin, the prognosis is more sinister, both as to suffering and duration of life. Any incidental complications will have to be taken into our reckoning, and these are far more perilous in the renal cases.

"Non-renal hyperpietics bear surgical operation fairly well; the renal very ill." [161]

The height of the arterial pressure reading is important, though an isolated reading has little more significance than an isolated record of temperature.

The actual pressure-reading on the sphygmomanometer is relatively of little account. What is important is the co-existence of symptoms and physical signs. A subject with a moderately raised arterial pressure may be dangerously ill, while another with a pressure which is excessively high may, nevertheless, have no symptoms and be able to do full work over a period of years.

Thus simple high pressure without unfavourable symptoms, even if it resists attempts at reduction, does not necessarily warrant a bad prognosis. If, however, the pressure rises rapidly or undergoes sudden elevations, the import is less favourable than when the pressure has mounted insensibly and keeps steadily at a fixed maximum. Even if the rise is permanent and associated with organic changes, life may possibly be continued for many years despite absence of treatment.

From time to time patients are seen whose pressure is continuously elevated apart from detectable cardiac, arterial or renal disease. Here the difficulty lies in excluding sclerosis of the cerebral, coronary or splanchnic vessels, since vascular changes may be very localised. Hence the value of expert ophthalmoscopic examination, which may reveal alterations in the fundus which are significant of arteriosclerosis.

Arterial pressure should not be considered as pathologically high unless the systolic pressure is *persistently* above 155 mm. Hg, and the diastolic *persistently* above

100 mm. Hg, but it should always be remembered that, as the result of increased peripheral resistance dependent upon tissue changes, arterial pressure rises more rapidly between sixty and eighty years of age. A moderate elevation of arterial pressure in a middle-aged patient may, *per se*, be disregarded.

TABLE IX

Pathologically High Arterial Pressures

	Systolic.	Diastolic.
Excessively high . .	280—320	165—180
Very high . . .	240—275	140—160
High	155—230	120—130
Suspiciously high if occurring before age 40 . .	145—150	90—110

Range of High Arterial Pressures

Dr. Maurice Campbell [162] has pointed out that there is a regular gradation in the relationship between increasingly high systolic and diastolic pressures. Starting from 160/100

TABLE X

Relationship between High Systolic and Diastolic Pressures

Average Standard.	
Systolic.	Diastolic.
160	100
180	110
200	120
220	130
240	140

for every 10 mm. rise in the diastolic, there is an increase of 20 mm. in the systolic.

Basal Metabolism in Hyperpietic Cases

In patients with hyperpiesia, whose renal function remains normal, the basal metabolic rate taken with Benedict's apparatus is usually normal, or occasionally shows a slight increase, the average being — 6. In most cases of hyperpiesis, however, in which renal function is damaged (primary and secondary contracted kidney), with systolic pressures ranging from 180 to 270 mm. Hg. and definite cardiac hypertrophy and dilatation, the basal metabolic rate is increased, the average being + 18.[163]

Importance of the Heart and Systolic Pressure in Prognosis

In prognosis the state of the heart is an important factor, although in many instances far too much influence is assigned to the heart and far too little to the arteries. The systolic pressure is an index of the maximum cardiac energy, though this may be profoundly modified by the pulse rate. In cases of permanently high pressure the onset of signs of commencing heart failure is of the gravest import. Such signs are dyspnœa, cyanosis, occasional or grouped premature contractions, gallop rhythm, total arrhythmia and continuously accelerated pulse rate. Any combination of these latter signs is indicative of cardiac defeat. "What usually happens is not simply the wearing out of the heart by excessive work, but the defeat of a labouring heart impaired by defective nutrition due to lesions of the coronary circulation, and deficient quality of blood resulting from damaged kidneys and other organs."[161] Evidences of the circulation becoming inefficient are afforded by notable deviation from the more normal 3 : 2 ½ ratio between systolic, diastolic and pulse pressures.

Arterial pressures may remain high during many years longout inducing cerebral hæmorrhage or anginal attacks, aboveven should these latter be severe or brought on by

exertion, the prognosis is far less grave than was formerly believed.[121]

The influence of heredity is important both in arteriosclerosis and in angina pectoris. In angina, if the pressure is high, the prognosis is better than if low, for in the latter event death often occurs unexpectedly. A rise in differential pressure, however produced, is always of much moment. Warfield[18] states that it is invariably accompanied by increase in size of the left ventricle, dilatation of the aortic arch and increase in the size of all the distributing arteries. The best examples of this syndrome are found in aortic regurgitation.

Importance of the Mean Pressure in Prognosis and Treatment

The mean pressure is a useful guide to prognosis and treatment.[58] A rise above previous levels solely of the mean pressure indicates the onset of high arterial pressure. In established cases, irrespectively of the height of the systolic pressure, the height of the mean pressure affords an indication of the gravity of the affection. So also, where symptoms are present apart from any material rise in systolic pressure, the existence of arterial supertension is suggested by the level of the mean pressure which is always above 120 mm. Hg., and prognosis is correspondingly less favourable. Increase of the mean pressure runs concurrently with increase of left venticular or total cardiac insufficiency. Latent insufficiency may be revealed by the amount and duration of rise of mean pressure consequent upon effort. Under normal conditions the mean pressure is not increased by effort.

Further, the mean pressure is useful as a guide to treatment. Cardiac tonics and rest lessen the mean pressure coincidently with amelioration in the symptoms and signs of cardiac insufficiency. When a drop has occurred, or the level remains stationary at a figure lower than it was before the institution of therapy, this may be discontinued. Failure to reduce the mean pressure indicates a bad outlook.